Acute Neuromuscular Responses to Resistance Training Performed at 100% of 10 RM and 90% of 10 RM

By

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Abstract

The purpose of the investigation was to determine the acute neuromuscular responses and total exercise volume of thirteen male subjects, experienced in resistance training, (RT) in response to two different RT protocols. Protocol A consisted of a single-arm flexion exercise for 3 sets of a 10 RM load to failure on each set using approximately 100% of a 10 RM. Protocol B involved 3 sets at 90% of a 10 RM load. Failure was determined by having subjects during protocol A perform each of the 3 sets at a 10 RM load until no further contractions could be completed. The subjects in protocol B preformed 10 repetitions for the first 2 sets at a 90% of 10 RM load. In the third set repetitions were performed until temporary muscle failure, regardless of the number of repetitions the subjects were able to complete. Both protocols incorporated a 3 min rest interval between sets. Blood lactate, MVIC, and EMG were measured prior to and upon completion of each loading protocol. When performing protocol B the subjects were able to complete a greater total volume (reps x sets x load) of exercise, but both protocols elicited similar significant increases in blood lactate and decreases in MVIC and $iEMG_{max}$ ($p=<.05$). Although, volitional fatigue was postponed while using 90% of 10 RM, the acute changes in neuromuscular fatigue were comparable, despite the 10% difference in loading intensity. Additionally, traditional strength training protocols involving repetitions to failure in each set may not optimize the total volume of resistance exercise, which may have implications for chronic adaptation.

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To all those who inhabited Barnes House, 6 Conroy Pl, and the Wickelow Palace in St John's, NF. Without those late night motivations and resultant early morning after effects, I could not enjoy sociable moments and still drive to the next watering hole. You helped provide me with the forum, time and freedom to create memories for the both of us. Often, I relied on your early morning reminders for the missing lines and clichés that culminated some of my most classic efforts – thank you.

Finally, I have to credit my Victoria influences. I acknowledge that Dotting the 'i' and crossing the 't' is not my strength and without your influence, I would not have completed this journey. Dr. Docherty and Dr. Gaul you were amazing mentors and consummate professionals. Dr. Bradenburg for his 'shaken and not stirred' efforts. and Bobby Cheema for providing me with the definition of 'moderately trained' and a plethora of bad newfie impressions.

Dedication

My entire Master's experience is dedicated to 2 special individuals and a length of tape that resided on the inside of a furnace closet in my Bishop's Falls, NF home.

From the time I was 10 yr old, dawning nothing more than hockey equipment and lofty goals I was told, "if you believe it, you will achieve it". The tape was lengthened to height of 6 ft. My parents made me believe that all I needed was a deep desire and firm believe. If those were in place, all else would take care of itself and, therefore the mark reached .

My dad and I took meticulous measurement of my progress for 7 yr. Well, at 5' 10" I never made that mark, but I did learn a valuable lesson – hard work and dedication will allow you to excel. To this day, when I have doubt or misdirected thoughts, I remind myself of the small gains and closer approximations to the end goal – 6' 0".

Mom, Dad, I owe this accomplishment to your belief in me!!

Introduction

The processes associated with fatigue have been empirically examined for decades, yet scientific understanding of how fatigue should be incorporated into resistance training is lacking. The concept of repetitions to failure during each training set, although widely accepted as necessary to optimize gains in strength and hypertrophy, has not been extensively studied. Failure during resistance training represents an inability to perform any further repetitions against a given dynamic constant external resistance (DCER) (Fleck and Kraemer, 1998). For example, an individual using 80% of 1 RM, reaches failure during a given set when the force producing capabilities of the muscles involved become reduced by 20%, and no further repetitions can be performed (MacDougall, Ray, Sale, McCartney, Lee, & Garner, 1999). Repetitions to failure represents a level of fatigue and that is relative to the percent of 1 RM being used. Repetitions to failure has been accepted as a method for inducing muscle adaptations even though controversy exists in regard to the mechanisms that underlie neuromuscular fatigue and subsequent muscle failure (Green, 1997).

It has been suggested that the design of the resistance training protocol may effect training specific muscle adaptations. Although, specific neuromuscular adaptations (myogenic versus neurogenic) are considered to be related to the manipulation, through the adjustment of the number of repetitions performed and the rest interval between multiple sets of an exercise within a given bout of DCER, the research supporting such relationships is equivocal (Kraemer & Fleck, 1998; Tan, 1999, Stone, Collins, Plisk, Haff, & Stone Stone, 2000). Stussi, Freitag, Hauestein, Wydler, Eigenmann and Boutellier (1998) and reviews by practitioners examining the practice of repetitions to failure during dynamic resistance training have challenged some of the

proposed relationships between training variables and specific neuromuscular adaptations (Baker, 1998; Kraemer, Duncan, & Volek, 1998; Stone, M., Chandler, Conley, Kramer, & Stone, E. 1996). Furthermore, regardless of the number of repetitions performed during a bout of resistance training and the rest between consecutive sets, it is believed that neuromuscular changes occur most readily with high, but sub-maximal loads to volitional fatigue (McCall, Byrnes, Fleck, Dickinson, & Kraemer, 1996; Staron, Karpondo, Kraemer, et al. 1994; Dons, Bollerup, Bonde–Peterson, & Hancke, 1979; Mcdonaugh & Davies, 1984). However, the contribution of muscle fatigue to muscle adaptations within these studies was not defined and is not well understood. It is possible that training protocols that increase the volume of work performed at an optimal intensity may effect the same or greater acute and chronic neuromuscular changes as protocols in which neuromuscular fatigue is induced for each training set.

The intensity of an exercise represents the amount of resistance used for a specific exercise and is usually related to the maximal load that can be lifted for a given number of repetitions within one set (RM), which is a common way to determine the load used during resistance training. Resistance training that maximizes both the volume (i.e. sets x reps x load) and intensity may be more important than inducing muscle failure on every set, particularly for increasing muscular strength and size. It has been suggested that the best way to optimize intensity and volume is to train at a percentage of an individual's true repetition maximum (Baker, 1998), permitting continued high intensity contractions without the drop-off in intensity or volume often observed during bouts of resistance training exercise designed to increase strength and hypertrophy (MacDougall et al. 1999). In spite of this, no evidence exists to support or refute this claim.

Some research has supported the use of repetitions to failure. Rooney, Herbert and Belnave (1994) found that temporary volitional fatigue protocols involving 10 RM were more effective in improving strength than training protocols that do not involve a temporary state of failure with the same load. It was speculated that during resistance training to failure, active motor units fatigue and their contribution to the generation of force is reduced. Consequently, motor units that were previously inactive are now recruited to assist in maintaining the force output. As a muscle group approaches temporary volitional failure, additional motor units are recruited, placing greater metabolic and neurological demands on an increasingly larger amount of muscle when compared to training that does not induce failure. Potentially, the increased demand would lead to greater muscle adaptation (Sale, 1988).

Research examining acute physiological changes within a muscle during sub maximal resistance loads via muscle substrate change and integrated electromyography (iEMG) have been equivocal and therefore must be examined further. Kulkulka and Clamann (1981) reported that iEMG readings in the adductor pollicis are near maximum levels at 55% 1RM and that most motor units were recruited by 30% of 1 RM, suggesting that most, if not all, motor units are recruited at much lower intensities than previously believed. However, the results also demonstrated that prolonged isometric actions at 50% MVC elicited a larger neural response in the biceps brachii, as measured by iEMG. The increases in iEMG were attributed to the recruitment of more muscle fibers as the duration of the muscle action continued. Furthermore, studies that examined selective fiber glycogen depletion (Robergs, Pearson, Costill et al., 1991; Pascoe, Costill, Fink, Robergs, & Zachweija, 1993) and synergist muscle activity (Behm, 1995) support the idea that increases in force generation at moderate to high relative intensities occur as a result of mechanisms other than increased motor unit recruitment.

Few studies have attempted to compare groups training to failure versus groups not training to failure (Berger & Hardage, 1967; Stowers 1993; Nimmons et al. 1995; Schott, McCully & Rutherford, 1995; Kraemer, 1997; Stussi et al. 1998; Sanborn, Boros, Hruby et al. 2000). Results from studies comparing failure to non-failure protocols have been relatively difficult to interpret, due to the different amounts of work used among the comparison groups (i.e. one set vs. multiple sets). However, Stussi et al. (1998) compared subjects who performed repetitions to failure at a 15 RM load to a group who performed 2 sets of 6 repetitions at a 10 RM load which did not induce failure, with a 2 min rest interval between sets. They found that the non-failure group increased their 1 RM similarly to the failure group. Although, only strength was measured, the volume and intensity used in the training protocols were similar to resistance training parameters currently considered to induce hypertrophy. However, although volume (i.e. total work) was equated as best as possible, the intensity between groups was varied. Additionally, this study did not involve measures of morphological change within the muscle, which prevents any conclusions regarding the effects of such DCER on muscle hypertrophy.

It seems likely that inducing muscle failure acts as a stimulus towards muscle adaptation (Rooney et al. 1994; Schott, McCully & Rutherford, 1995). However, the degree to which muscle failure, induced by moderate to high submaximal loads involving DCER protocols, effects acute neuromuscular change has not been carefully examined. Furthermore, previous studies have not determined how optimizing volume through manipulating intensity effects acute performance over repetitive sets of DCER. Baker (1998) has proposed that training to failure on repetitive sets prevents optimization of total work volume for any muscle used during resistance exercise, which ultimately would decrease the acute neuromuscular stimulus and chronic training adaptation. He suggested that training at 90% of the selected repetition maximum optimizes the

capability of the muscle to perform a high volume of work over 3 sets of resistance training. According to Baker (1998) the increase in total work performed would provide a more powerful stimulus for adaptation. Although Baker (1998) suggests that total training volume may be linked to neuromuscular adaptations resulting from resistance training, examination of acute physiological responses that result from working at 100% of 10 RM and 90% of 10 RM must preclude examination of chronic neuromuscular adaptation. In order to test the hypothesis of Baker (1998), the acute neuromuscular training responses of resistance trained athletes training at 100% of 10 RM and 90% of 10 RM for each training set were measured. It was hypothesized that both protocols would induce analogous acute neuromuscular responses at the completion of the 3 sets but training volume would be greater in the 90% of 10 RM protocol.

Purpose

Previous research has not examined the acute neural and metabolic physiological changes associated with changes in the percentage of 10 RM effort during RT designed to produce cumulative fatigue. The purpose of this investigation was to determine the acute blood lactate and iEMG responses of two different RT protocols that induced acute fatigue. A second purpose was to determine whether the total work or volume achieved over three sets would be different when trained individuals utilize loads representing 90% of a 10 RM compared to when they trained at 100% of 10 RM (i.e. repetitions to failure) on each set.

Research Questions

Models of the development of muscular strength and hypertrophy suggest that high volume, short rest and low to moderate intensity induces the greatest increases in muscle strength

and hypertrophy. The acute production of lactic acid has been previously related to a reduction in force output over 3 sets of 10 repetitions (MacDougall et al., 1999). Muscle lactate levels were found to be higher following 3 sets of 10 repetitions when compared with 3-5 sets of 5 repetitions, with similar 3 min rest between sets (Gordon, Kraemer, Vos, Lynch, & Knuttgen, 1994). However, recent research investigating the role of fatigue in muscle adaptation is equivocal. The popular practice of "repetitions to failure", frequently used by individuals attempting to induce muscular hypertrophy, has been supported by Schott, McCully and Rutherford, (1995) and Rooney et al. (1994) but a recent investigation comparing repetitions to failure with repetitions not to failure found that failure produced no adaptive advantage (Stussi et al., 1998). These recent findings have led to the following questions:

1. Are acute blood lactate and iEMG levels induced through 100% of 10RM with 3 min rest similar to acute lactic acid and iEMG levels which result from training involving 90 % of 10 RM training with the same rest and repetition protocol?
2. Can individuals working at 90% of 10 RM, as proposed by Baker (1998), perform a greater total volume of work over three sets of resistance training than individuals working at 100% of 10 RM?
3. Are the changes in maximal force similar when working at 90 % of 10 RM and 100% of 10 RM for three consecutive sets?

Significance of the study

This study may have important ramifications for the way in which resistance training is prescribed to resistance trained athletes, provided that similar acute responses reflect chronic

training effects. It is a currently accepted practice that training to failure on repetitive sets is necessary to induce the largest gains in strength and hypertrophy. However, it is possible that the current exercise protocols are taxing physical and mental reserves of athletes more than is necessary or optimal. If the contentions regarding acute neuromuscular responses of Baker are shown to be correct, well-trained individuals may experience chronic benefits from training with less fatiguing resistance exercise protocols, leaving them better prepared for practice and competition.

Methodology

Subjects

Subject characteristics are outlined in Table 1. 13 college-aged males, with at least 1 yr weight training experience and who primarily utilized bodybuilding/hypertrophy type loading protocols, volunteered for the study and completed 4 sessions which included familiarization, testing and the two resistance training protocols. The subjects had probably achieved significant neuromuscular adaptations due to their previous training history which would have reduced the possibility of any training effects during the study that may have affected the results. Baker (1998) described each repetition for the beginner as "a trial for learning", suggesting that with beginners, even a single training session could lead to increased neural drive. If learning occurs so readily, a reduction in relative intensity could occur within a single resistance session. In addition, trained subjects are better prepared to tolerate the metabolic demands of the 10 RM loads used in this study which was shown to be a concern in prior investigations (McCall, Byrnes, Fleck, Dickinson, & Kraemer, 1999). Two sessions were used for familiarization of the

experimental protocols, collection of descriptive variables, and verification of 10 RM for unilateral forearm flexion using the biceps preacher curl exercise.

Table1. Subject characteristics. Values are means and SD.

Age (yr)	25.5	0.3
Height (cm)	176.5	5.8
Body mass (kg)	84.5	6.3
Training status	min. 1 yr	N/A

All procedures were approved by the University of Victoria ethics review board and were explained to subjects prior to testing. Written informed consent was obtained from all participants prior to the beginning of experimental testing.

Procedures

Training session

Both protocols are outlined in figure 1. Each of the 100% of 10 RM and 90% of 10 RM protocols were performed in random order to prevent any effects that may have occurred due to the order of the conditions. A typical day of testing is outlined in Fig. 2. This design allowed for a quantitative approach, permitting examination of responses to moderate resistance exercise based on the specific changes to the exercise protocol. A moderate-volume training regimen, which has been previously shown to produce significant acute increases in exercise induced blood lactate levels (Kraemer et al., 1990; McCall et al., 1999), was used for the failure condition. The 90% of 10 RM condition involved a protocol designed to permit subjects to perform the same number of repetitions on each set. It was proposed that temporary volitional failure would occur only on the last set when using this protocol. Regardless if this occurred on the 10^{th} repetition, during the third set repetitions were performed until temporary muscle failure, regardless of the number of repetitions the subjects were able to complete.

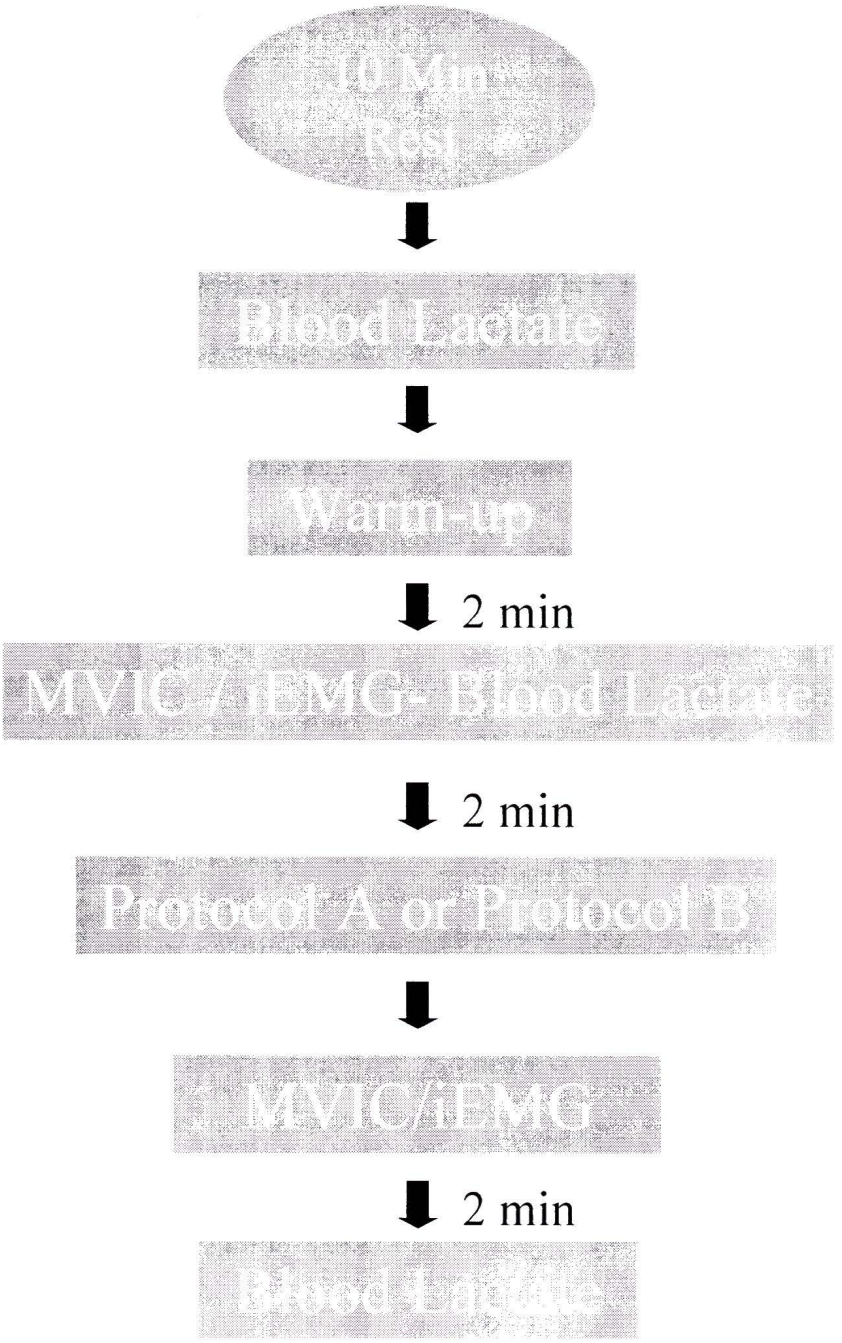

Figure 1. Experimental design consisting of either the 90% of 10 RM or 100% of 10 RM protocol.

Strength testing

All subjects were assessed for initial strength while performing unilateral forearm flexion on a preacher curl bench. The testing session occurred at least 72 hr prior to any experimental trials. Upon arrival, subjects were required to progress through a warm-up protocol, involving one set of 10 repetitions at approximately 30-40% of the predicted 1 RM, and two sets of two repetitions representing approximately 60% and 80% of 1 RM. Each warm-up set was followed by at least 2 min rest. The warm-up was designed to minimize metabolic demand on the muscle. Additional sets of 2 repetitions were used if subjects did not feel sufficiently prepared to perform a 1 RM test. Testing always involved performing the 1 RM prior to 10 RM, and 4 min rest intervals were used between testing sets. If the weight selected for the first RM testing could be moved for more than the required number of repetitions subjects were encouraged to perform repetitions to failure in an attempt to find or better estimate his 10 RM on the subsequent test trials. In the case of a failed repetition, the weight was removed from the subjects hand, a 2 min rest administered, and resistance adjusted according to the researchers best estimate. If subjects were able to perform more or less than the desired repetitions, but were within one repetition of the goal on the 10 RM trials, the predictive charts of Bompa (1998) were used to determine the resistance needed to perform 10 repetitions maximum.

Experimental condition

Upon arriving at the laboratory, subjects were given 10 min of seated rest, facilitating the clearance of any lactic acid that may have resulted from any activities performed by the subjects prior to arriving at the laboratory. The order of events is shown in Fig. 1. Blood lactate was measured immediately after the 10 min rest. Blood lactate measurement was followed by the

warm-up protocol. The warm-up involved the exact same protocol as during the testing with the following exceptions. Two minutes after the warm-up protocol a maximal isometric action was performed. Immediately after the first maximal isometric action a finger prick blood lactate measurement was taken to ensure that the isometric action did not cause elevated blood lactate prior to the experimental protocols. Upon completion of the warm-up, the experimental protocol was initiated.

Dynamic muscle contractions

A supported, dumbbell forearm flexion exercise was performed using the subjects non-dominant arm on a preacher curl bench. A metronome was set to 40 beats per minute to assist in the timing of forearm flexion. The eccentric and concentric components of the exercise were both performed on a 1.5 s count.

The supported forearm flexion involved a muscle mass which has been previously shown to induce reliable increases in muscle and blood lactate (MacDougall et al., 1999). The exercise was structured so that the angle at the elbow and range of motion were noted and kept constant for each set of the prescribed exercise sessions. During forearm flexion/extension, the elbows of both arms were placed on the angled pad, such that the posterior upper arm rested flat against the pad; the back was held in an upright position; the feet remained flat and stable and the axillary region was not permitted to rest over the edge of the bench. The latter procedure minimized brachial occlusion. No gripping with the inactive arm occurred as the arm hung freely over the front of the bench with palm supinated. This seating position minimized trunk rotation and maximized the contribution of the biceps brachii in the action.

When performing protocol B were instructed to perform 10 consecutive repetitions at the required intensity. For protocol A, subjects were instructed to perform as many repetitions as possible until no further muscle actions could be performed at the 10 RM load. If subjects performing protocol A were able to perform more or less than the required number of repetitions on set 1, the set was continued until temporary volitional fatigue occurred. If the number of repetitions completed was not within 2 repetitions 10 RM, then the session was terminated, weight charts of Bompa (1998) used to adjust the resistance settings for future testing, and the subject was asked to return to repeat this condition after a minimal of 72 hr. If subjects performed no less than 8 and no more than 12 repetitions, the tester recorded the data and continued the session. The number of repetitions at muscle failure was recorded. If failure to complete all 10 repetitions occurred during protocol B, the tester would have followed the procedure outlined for the 100% of 10 RM protocol (i.e terminated the session and adjusted the weight for another session performed at least 72 hr later).

Maximum Voluntary Isometric Actions

All isometric actions were performed on a Cybex II isokinetic dynamometer at an elbow angle of 90°. Subjects were directed to contract as fast and as forcefully as possible and hold all contractions for 2-3 s. Maximal force and iEMG were simultaneously measured. Force outputs were stored on a computer interfaced with the isokinetic dynamometer.

iEMG

Silver chloride electrode leads were applied to the belly of the bicep brachii muscle on each subject's dominant side. A 10-15 mm distance and a bipolar configuration between the

centers of electrodes were used to help minimize cross-talk amongst synergistic and antagonistic muscle groups. The electrode placement areas were scrubbed of dead skin cells with an alcohol soaked pad prior to electrode application. Electrodes were attached with double adhesive bands and conductive gel placed in the center region of the electrode. Ground electrodes were then placed on the styloid process of the wrist. Peak iEMG amplitudes and $iEMG_{mean}$ were recorded during MVIC. Measurement began .25 s after the beginning of the MVIC and lasted for 1 s and all information was stored to Biopac Systems, AcqKnowledge III. All EMG signals were rectified and integrated. The preamplifiers were interfaced with a Pentium II PC. Following raw data collection, all data were stored and later analyzed using the AcqKnowledge III EMG software package.The EMG signal was quantified by two methods: integrated EMG (area under the curve) and peak EMG muscle activity during the performance of each activity.

Lactate Measurement

Finger prick blood samples were taken (<5 µl/sample) for determination of peripheral lactate concentrations from the middle digit of the non-exercising arm. Lactate and iEMG readings were obtained following the procedures of Kraemer et al. (1990) and are illustrated in Figure 2. Blood samples were drawn prior to warm-up, 2 min after warm-up and throughout resistance exercise as illustrated.

Experimental design

Each subject performed both protocols. A random and balanced crossover design was used, ensuring that each subject had an equal opportunity to begin testing with either the failure or non-failure protocol. At least 72 hr was allowed between each training/testing session. Between sessions subjects were encouraged to avoid all forearm flexors resistance training for 72

hr and all resistance exercises where the forearm flexors acted as a secondary mover (i.e. back exercises) for 48 hr prior to the next training/testing testing.

All subjects were asked to refrain from alcohol and caffeine consumption for a period of 8 hr prior to testing. Subjects reported to the laboratory following at least 2 hr of fasting. Upon reporting to the laboratory, each subject was permitted to drink 500 ml of water prior to exercise in order to standardize the level of hydration (Gotstalk et al., 1997). At that time an EMG electrode was placed on the motor point of the biceps brachii. Subjects then rested for 10 min prior to the actual data collection to minimize hormonal and blood lactate fluctuations related to prior activity and anticipatory responses (Kraemer et al., 1991).

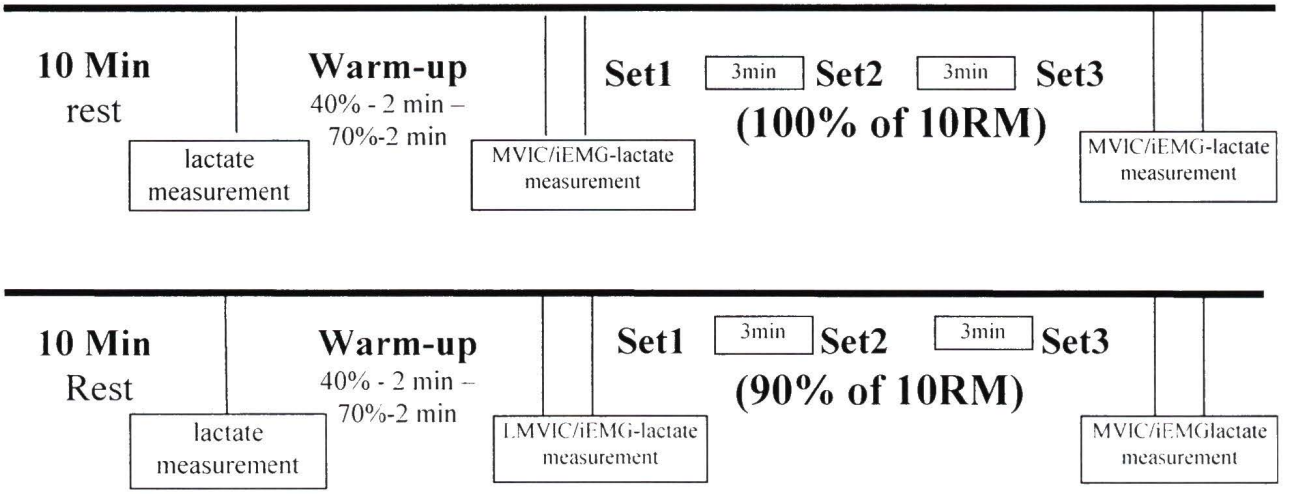

Figure 2. Random ordered experimental design, including order for bicep brachii iEMG recording and blood sampling for resting and exercise sessions.

In order to ensure that the warm-up and MVIC did not affect pre exercise blood lactate, finger prick samples were obtained following the same procedures as the resting conditions, except the subjects performed a maximum voluntary isometric action immediately prior to sampling. Subjects were placed in a seated position for sampling at pre and post exercise. Post exercise sampling occurred at 2 min. Water was available ad libitum during exercise and recovery. All blood samples were immediately placed into a YSI lactate Analyzer, Yellow Springs, OH. The testing protocol allowed for all samples to be determined within the testing session.

Data Treatment and Statistical Analysis

Paired sample t-tests were performed on forearm flexion for each of the dependent measures (i.e. total volume, blood lactate, $iEMG_{max}$, $iEMG_{mean}$ and MVIC). Pre to post effects were examined within protocols; pre exercise to post exercise effects were examined between protocols and to ensure no pre-test differences between exercise conditions, pre exercise values were compared. For all t-test groups the training protocol served as the independent measure. Volume, blood lactate, iEMG, and MVC were the dependent measures and are presented in Table 1. Correlations were conducted between protocols for pre exercise and post exercise means, for each of the dependent measures and are presented in Table 2. Planned comparisons include:

1. H1: Protocol B > Protocol A (volume of work)
2. H2: Protocol B = Protocol A (Blood lactate, MVC and iEMG)

All values were averaged across group and analyzed with SPSS 9.0. Integrated EMG and MVIC were used as indicators of decreases in force during and across sets. Significance was set at the $p \leq .05$.

Results

13 subjects performed forearm flexion using two different exercise protocols. During both sessions total volume (reps x sets x load), maximum voluntary isometric contraction (MVIC), mean area under the curve ($iEMG_{mean}$) and peak iEMG amplitude ($iEMG_{max}$) values were obtained pre and post exercise- for forearm flexion at 90% of 10 RM and 100% of 10 RM. The mean 10 RM for the subjects was 76.6% (4.2 %) of 1 RM (Table. 1).

Volume

There was a significant 14% difference between the two protocols for the mean training volume performed across three sets ($t_{1, 12} = 4.02$; $p = 0.002$), with the subjects following protocol B showing a greater volume of exercise compared to protocol A over 3 sets (Fig. 3).

MVIC

A significant 19% for protocol A and 18% for protocol B within protocol decrease was observed pre- to post- exercise for mean MVIC, ($t_{1, 12} = 9.87$; $p = 0.001$, $t_{12} = 11.49$; $p = 0.000$ for protocols A and B respectively), but no statistical difference was observed between exercise protocols (Fig. 4).

$iEMG_{mean}$

There was a significant difference in $iEMG_{mean}$ between protocols pre - to pre - ($t_{1,12}$ = 3.23; p = 0.03), with protocol A having a lower mean value than protocol B. There were no significant differences observed within protocols ($t_{1,12}$ = 0.30; p = .44, for protocol A), although protocol B approached significance ($t_{1,12}$ = 2.06; p 0.051). No between protocol differences were observed pre - to post - for mean iEMG area under the curve (Fig. 5).

$iEMG_{max}$

No statistical difference was found between the two exercise protocols for $iEMG_{max}$. However, a trend towards between protocol decreases for $iEMG_{max}$ ($t_{1,12}$ = - 1.67; p = 0.67) was observed post exercise with significant decreases from pre - to post - exercise for both protocols ($t_{1,12}$ =2.33; p = 0.38 for protocol A and t_{12} = 5.62; p = 0.000 for protocol B) (Fig. 6).

Blood Lactate

Significant differences in mean blood lactate were observed within each protocol ($t_{1,12}$ = 8.31; p < .05, $t_{1,12}$ = 4.96; p = 0.000 for protocols A and B, respectively), but not between protocols for blood lactate from pre - to post - exercise (Fig. 7).

Mean Number of Repetitions

A significant drop off in the mean number of repetitions performed on set 2 was observed ($t_{1,12}$ = 5.78, p <.000). Similarly, after the completion of 3 sets significant mean differences were observed for subjects performing protocol A (6.07 ± 1.55), but no

significant decrease occurred for protocol B (10.54 ± 1.66) (Fig. 8). Significant between group differences were observed, with protocol A resulting a greater drop-off in repetitions than protocol B ($t_{1,12} = -7.493$, $p < .000$) (fig. 8).

Correlations for Dependent Measures

A significant post exercise relationship between blood lactate and $iEMG_{mean}$ was found between protocols ($r_{1,12} = .572$, $p < .041$), but no relationships were found post exercise for any other statistically compared variables (Table 3.). All other relationships are listed in Appendix C.

Table 2. Mean (SD) total volume, total repetitions, repetitions per set, MVIC, $iEMG_{mean}$, $iEMG_{max}$ and blood lactate after the performance of 3 sets of forearm flexion at 100% of 10 RM (A) and 90% of 10 RM (B).

Training Variables	p-value (between protocols)	Protocol A (100% of 10 RM)	Protocol B (90% of 10 RM)
Total Volume (lbs)	0.002	901.72(115.58)*	1028.61(147.74)**
Repetitions on set 3	< 0.000	6.07(1.55)**	10.54(1.66)
MVIC (Nm)			
Pre	.923	88.85(9.83)	89.20(9.40)
Post	.454	71.8(10.39)*	73.52(11.66)*
$iEMG_{mean}$ (uV)			
Pre	.007	0.21(.13)	0.40(.27)***
Post	.220	0.19(.15)	0.27(.22)
$iEMG_{max}$ (uV)			
Pre	0.09	2.62(.94)	3.59(2.12)
Post	0.12	2.18(1.35)*	2.94(1.85)*
Blood lactates ($mmol \cdot L^{-1}$)			
Pre	0.580	1.34(.40)	1.74(.63)
Post	0.995	2.92(.68)*	2.76(.76)*

* Represents significant within protocol pre-to post exercise differences
** Represents significant between protocol post exercise differences
*** Represents significant between protocol pre exercise differences

Table 2. Correlations matrix for post MVIC, blood lacate, $iEMG_{mean}$, $iEMG_{max}$ after 3 sets of forearm flexion at 100% of 10 RM (A) and 90% of 10 RM (B).

Protocol A	Blood Lactate	MVIC	$iEMG_{mean}$	$iEMG_{max}$
Blood Lactate	1.00	-.223	.572*	.220
MVIC	-.223	1.00	.933	.513
$iEMG_{mean}$	.572*	.132	1.00	.624*
$iEMG_{max}$	.220	.513	.624*	1.00
Protocol B	**Blood Lactate**	**MVIC**	**$iEMG_{mean}$**	**$iEMG_{max}$**
Blood Lactate	1.00	.026	-.274	-.355
MVIC	.026	1.00	.263	.198
$iEMG_{mean}$	-.274	.263	1.00	.906*
$iEMG_{max}$	-.355	.198	.906*	1.00

* Represents significant between group differences from post - to post -

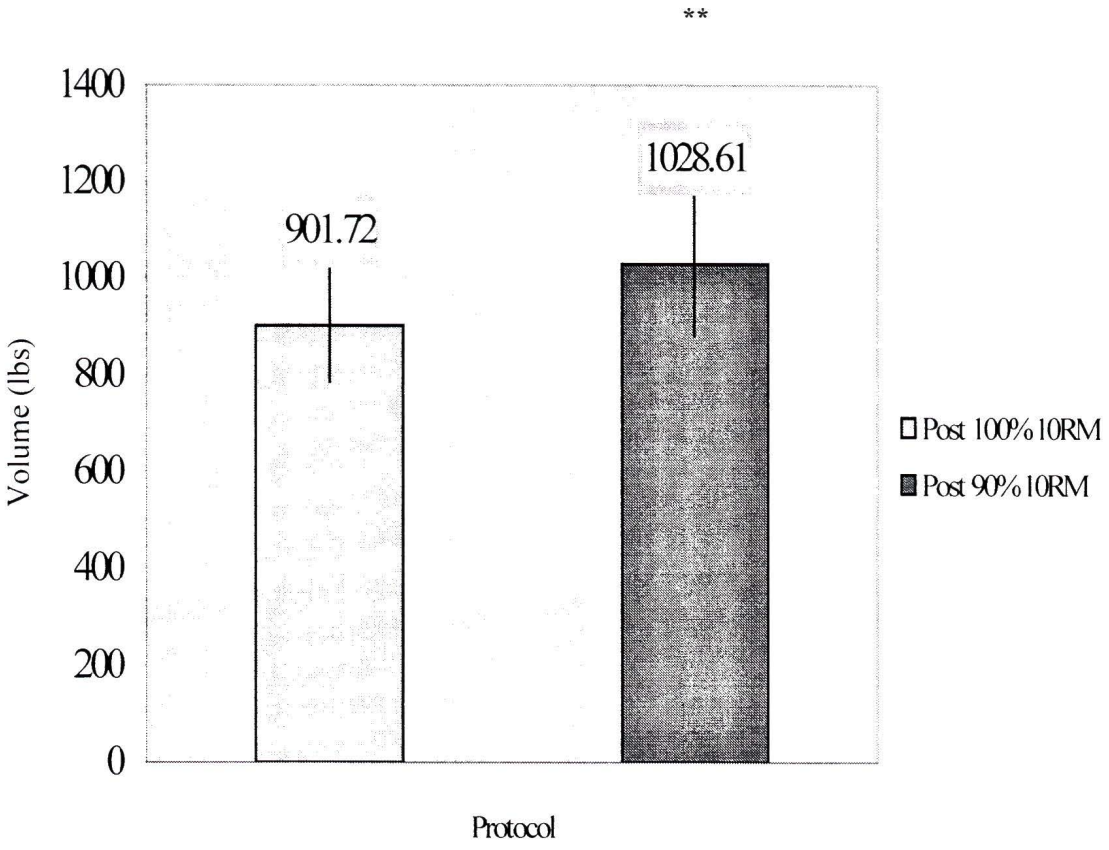

Figure 3. Mean (SD) Total volume from performing 3 sets of forearm flexion for each protocol. Volume = (sets x reps x load) for forearm flexion at 100%(A) or 90%(B) of 10RM.

** Represents a significant difference between protocols from pre - to post - exercise

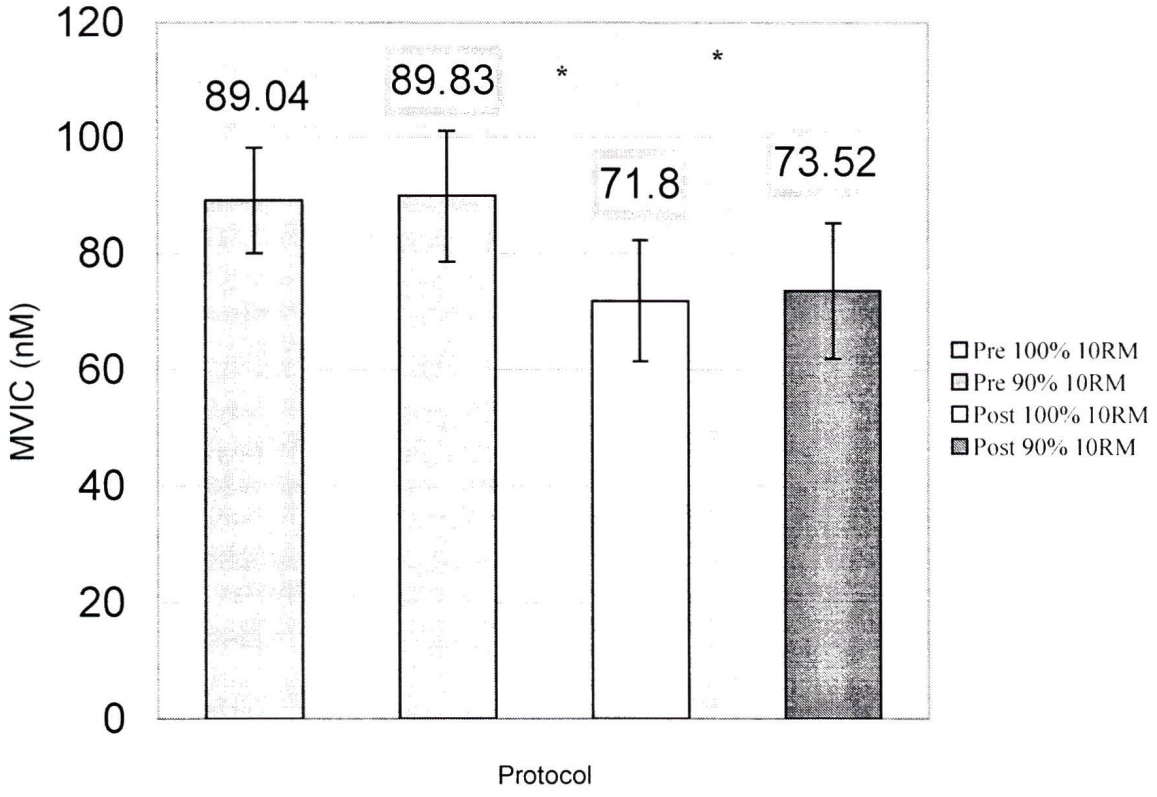

Figure 4. Mean (SD) maximum isometric contractions produced during voluntary single forearm flexion pre- and post- at 90 or 100% of 10 RM

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*Represents a significant difference within protocol from pre - post exercise

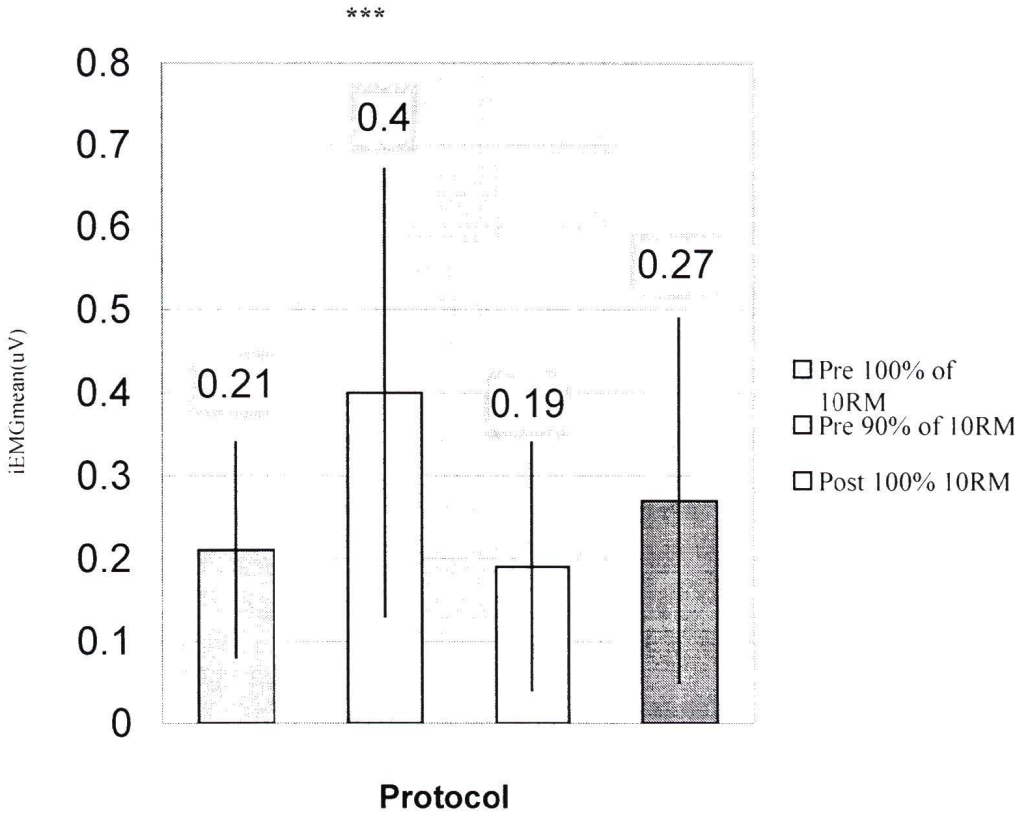

Figure 5. Mean integrated area under the curve produced during single arm forearm flexion pre- and post- training performed at 90 or 100% of 10RM

*** Represents significant pre exercise differences between protocols

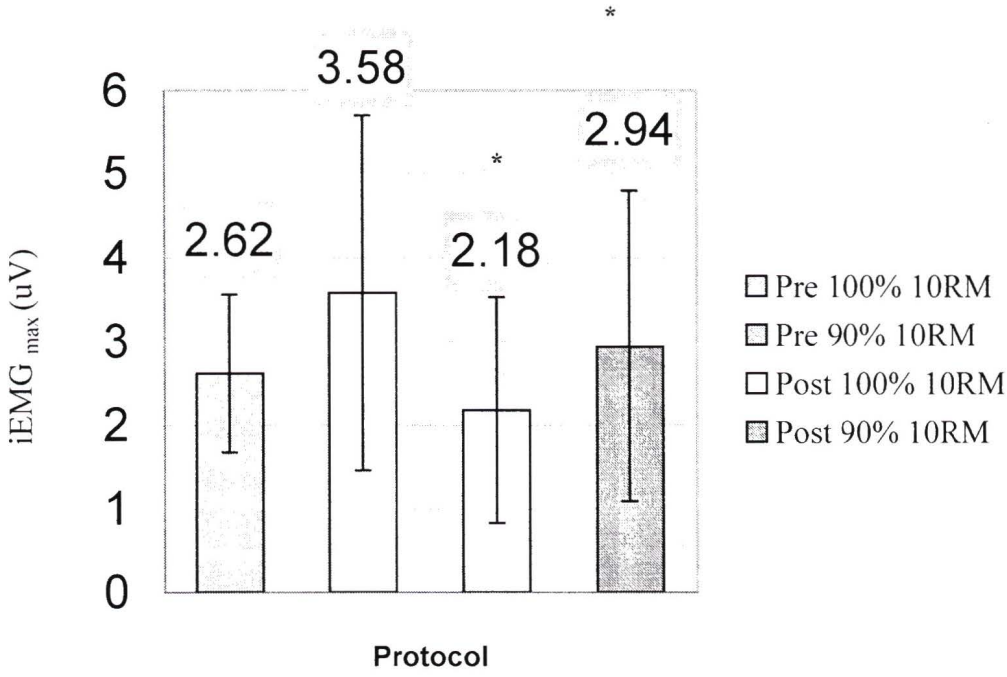

Figure 6. Mean (SD) values of iEMGmax for single arm foerarm flexion pre -and post- training performed at 90 and 100% of 10RM.

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* Represents a significant difference within protocols from pre - to post - exercise

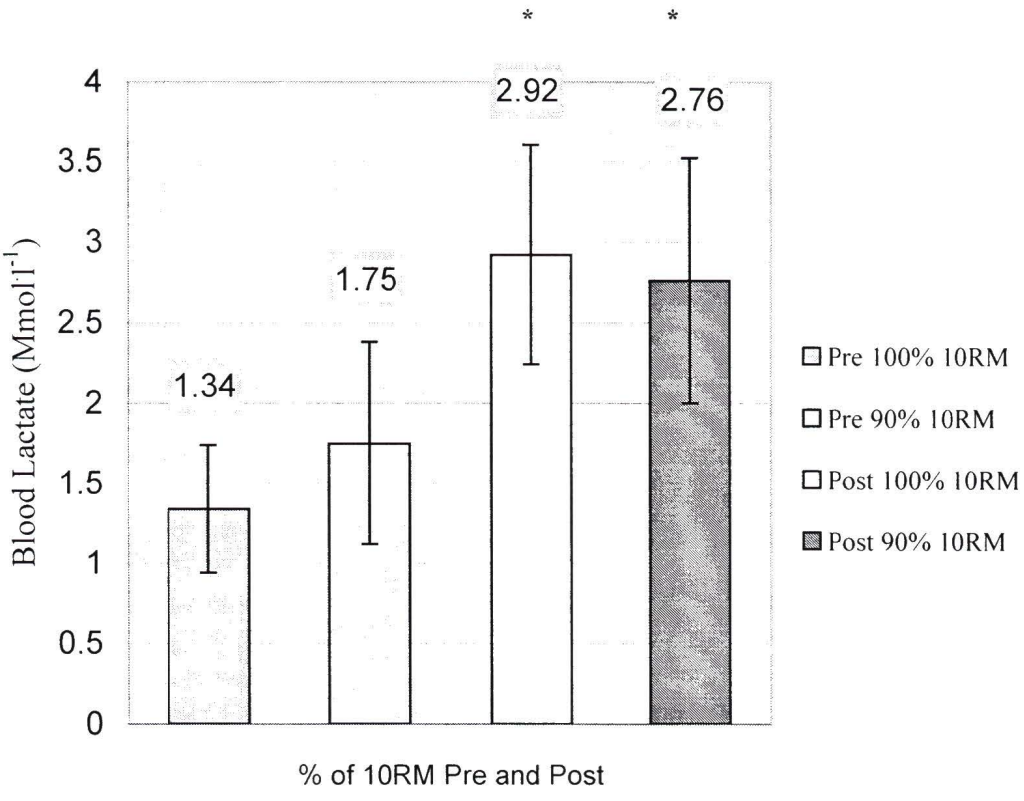

Figure 7. Mean (SD) blood lactate levels recorded pre- and post- training of forearm flexion performed at 90 and 100% of 10RM.

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*Represents a significant difference within protocols from pre - to post - exercise

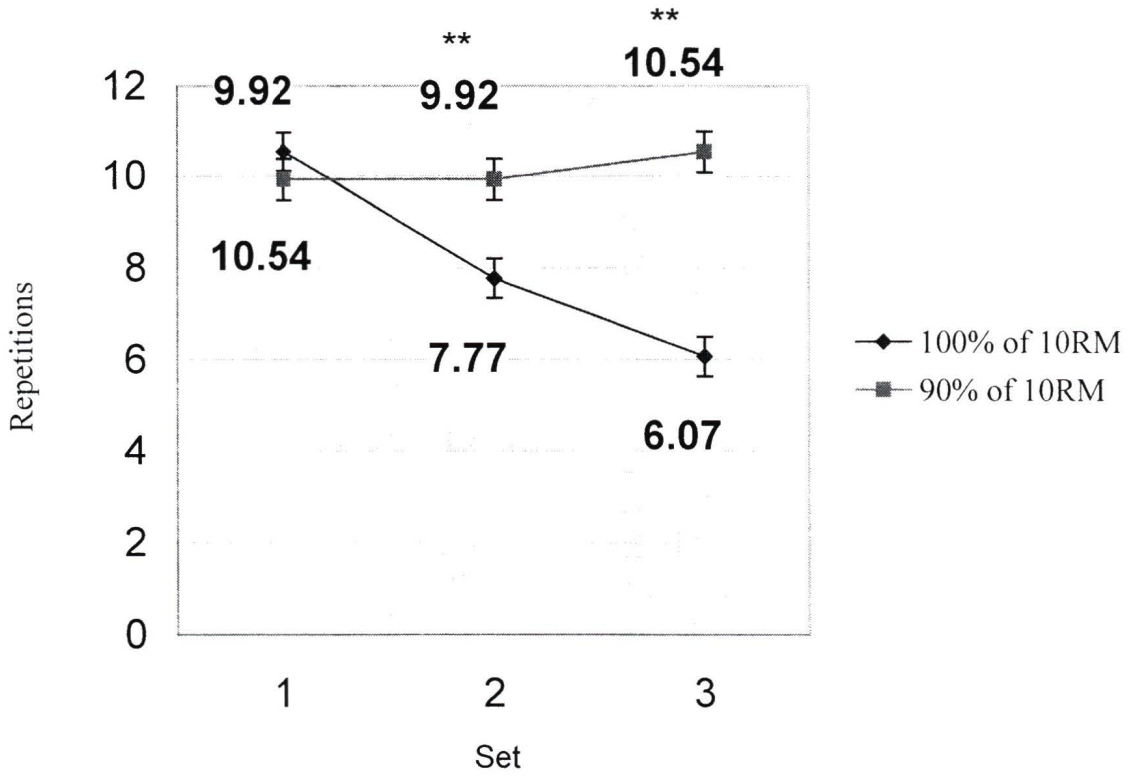

Figure 8. Mean (SD) number of repetitions completed for each set while performing single arm forearm flexion at 90 or 100% of 10RM.

** Represents significant between protocols differences after the completion of set 2 and 3.

Discussion

The purpose of this investigation was to examine and compare acute neuromuscular fatigue produced by 2 resistance training protocols: A) 3 sets of single-arm forearm flexion until failure utilizing a 10RM load and B) 3 sets of single arm elbow flexion at 90% of a 10 RM load.

Volume

The results confirmed the hypothesis of Baker (1998) that traditional, 3 sets of 10 RM resistance training strategies do not maximize the total volume of work performed during an acute bout of forearm flexion. The total volume of work (i.e. volume load) was significantly reduced when acute fatigue was induced on repetitive sets of 10 RM training with 3 min of rest between each set (Fig. 3). Specifically, the 90% of 10 RM protocol resulted in a 12.3% greater volume of exercise than the 100% of 10 RM group after the completion of all 3 sets. The differences in volume can best be explained by the drop-off in repetitions performed during protocol A. The present findings are in agreement with previous research that has shown 3 min is an insufficient amount of interset recovery time in resistance training when the previous set is performed to volitional muscle fatigue (Pincevero, Lephart, & Karunakara,1997; 1998; MacDougall et al., 1999; Esposito, Orizio, & Veicsteinas, 1998).

MacDougall et al. (1999) found that 3 min rest between sets at 12 RM was insufficient to allow full recovery of the forearm flexors muscle. The inability to recover within the experimental rest interval resulted in a "drop-off" of repetitions performed in the second and third sets. Average repetitions completed per set decreased from 11.7, 9.2, 7.2 in the consecutive sets. These results were similar to those obtained in the present study. Subjects performing

protocol A completed 10.54 (1.45), 7.77 (1.24), 6.07 (1.55) on sets 1, 2 & 3 respectively (fig. 8). Furthermore, the number of repetitions completed during protocol A were significantly lower on set 2 (9.92 versus 7.77) and set 3 (6.07 versus 10.54) than when subjects performed forearm flexion at 90% of 10 RM. These results suggest that the higher volume experienced by the 90% of 10 RM protocol was a result of the ability of the forearm flexors to maintain the volume load per set. This difference was observed even though subjects working at 90% of 10 RM began each set with a lower load than that used in protocol A. Similar drop-offs have been observed in the quadriceps (Pincivero et al., 1998), when researchers utilized a similar rest interval and intensity as the current study. However, direct comparison is difficult because the quadriceps is a larger muscle group with a different fiber composition compared to the forearm flexors.

Recovery from repetitions to failure of moderate to high intensity exercise has been shown to extend well past 3 min (Pincervo et al., 1997; Esposito et al., 1998; MacDougall et al., 1999). Esposito et al. found a 26% decrease in MVIC after 10 min of recovery from two sustained isometric actions of the forearm flexors to volitional failure at 80% and 50% of MVC. The total time under tension for test 1, performed at 80% of MVC lasted an average of 25 s, which was very similar to the total time under tension for set 2 at 80% (24 s) in the present study. No neuromuscular explanation for this extended recovery was given, but it might be speculated that the fast twitch glycolytic motor units were derecruited as result of fatigue and were unavailable for recruitment during subsequent sustained contractions. Alternatively, it could be concluded, based on the similar durations and intensities that a similar motor unit pool was available and other mechanisms, perhaps distal to the neuromuscular junction, were responsible for the acute neuromuscular fatigue during tests 1 and 2. This peripheral fatigue could slow cross

bridge cycling (Vestergaard-Poulson, Thomsen & Sinkjaer, 1993) which would lead to a decrease in force producing capabilities of the muscle.

Subjects in the present study were working at approximately 76% of 1RM during the first set to failure in the 100% of 10RM group. One explanation for the drop-off might be related to changes in available muscle glycogen. MacDougall et al. (1999) observed a 24% decrease in muscle glycogen and progressive reduction in the rate of glycogenolysis over three sets of forearm flexion. Given the inability of the forearm flexors to supply energy at a sufficient rate, the force producing capabilities of the forearm flexors would be reduced. Furthermore, it is possible that when a muscle is not taken to failure during a bout of resistance exercise that the rate of glycogenolysis may be maintained at a higher level than subjects performing sets to failure, allowing the muscle to supply muscle glycogen faster for subsequent sets. Although, the rate of glycogenolysis was not tested in this study, when a muscle is taken to failure, the change in glycogenolysis may follow a non-linear relationship (McCartney, Spriet, Heigenhauser et al., 1986; Robergs et al., 1993), such that subsequent sets to volitional fatigue lead to smaller reductions in the rate of glycogenolysis. The slowing rate of change suggests that a limit exists for ATP and glycogen utilization preventing repetitions to failure during resistance training from exhausting existing stores.

Support for the metabolic ceiling, as seen with slowed glycogenolysis, can be found in studies that have examined high intensity anaerobic sprinting (McCartney et al., 1986; Vestergaard-Poulson, Thomsen, Sinjaer, & Henriksen, 1995; Bogdanis et al., 1996). Many similarities exist between all out efforts on the cycle ergometer and moderate resistance training for the lower body, making cycle ergometer studies a useful model for comparison regarding the rate of glycogenolysis. Vestergaard-Poulson et al. (1995) found that a metabolic limit existed

during anaerobic activity for reactions involving excitation-coupling at the sarcoplasmic reticulum, limiting the ability of the muscle to further produce substrates, and ultimately force, through reduced muscle contractility in an anaerobic state once the 'limit' had been reached. Further support comes from research on ATP utilization, where it has been observed that ATP is not significantly altered during repeated bouts of high intensity intermittent exercise (Bogdanis, Nevill, Lakomy, & Boobis, 1996; 98). It was suggested that a metabolic ceiling exists to prevent further splitting of Pi from ATP. Because all energy systems of the body are linked, it follows that glycogenolysis would also be slowed, preventing a stressed muscle from utilizing all of its available energy. In fact, Bogdanis et al., using 30 s all out sprints on a bicycle ergometer, found that aerobic metabolism supplied 49% of the energy during sprint 2 and that oxygen uptake increased from 2.68 to 3.17 $L.\bullet min^{-1}$ between sprint 1 and sprint 2, respectively. Furthermore, ATP utilization was asymptotic, suggesting an upper limit to the contributions from the anaerobic system. Following repeated bouts of maximal cycle ergometry, McCartney et al. (1986) examined glycogenolysis and found that both the rate of glycogenolysis and muscle lactate levels did not change on the third and fourth bouts.

These findings suggest that when lactate levels reach a level where production exceeds utilization, consequent changes in hydrogen ion and inorganic phosphate production may inhibit maximal force production. Decreases in the rate of glycogenolysis may lead to or be the result of a fatigue-induced state that is indicative of inadequate energy supply. If the supply of ATP, whether aerobic or anaerobic, is not sufficient to meet the current demand, a disproportionate reduction in the force producing capabilities of the muscle will occur.

Given that type IIb fibers have the largest quantities of ATPase (Staron et al., 1994), it is important from a protective point of view, that type IIb fibers are derecruited via feedback,

preventing depletion of the limited pool of ATP, leaving only the more oxidative type IIa and type I fibers. Needed ATP could be produced through aerobic metabolism (Bogdanis et al., 1996), but at a reduced rate compared to anaerobic mechanisms. However, not all results have confirmed increasing contributions of aerobic metabolism (Bogdanis et al., 1998). Furthermore, if a shift towards oxidative fiber utilization (type I and type IIa) with increased mitochondrial content did occur, the rate of metabolite production would be reduced. Fibers with oxidative capabilities produce less force, possibly contributing to an inability to perform as many repetitions during high intensity resistance training on subsequent sets of forearm flexion performed at the same intensity. Metabolites are consequently produced at a lower rate, slowing metabolite accumulation. If an upper limit to metabolite removal exists and there is feedback to the neuromuscular system via the periphery, it would seem likely that the muscle would decrease ATP degradation until the metabolite levels returned to this "fail safe" level (Gandevia, 1992). The results of the current study lend support to this theory. If a "ceiling" exists, cumulative blood lactate response would be expected to be similar at post exercise for both protocols. Because no statistical difference was found pre - to post - forearm flexion between protocols A and B, it might be speculated that a metabolic limit exists and was obtained during the current protocols.

From these results it might be speculated that the rate of anaerobic metabolism decreased during the current study. Examination of the number of repetitions performed across sets revealed a change in the percentage drop-off in repetitions across sets during the 100% of 10 RM protocol. The percent change in repetitions performed from set 1 to 2 was 26.3%, whereas from set 2 to 3 the drop-off was 21.9%. The drop-off in repetitions within protocol A and subsequent reduction in volume accrued in the 100% of 10 RM may be the result of large peripheral

demands involving muscle metabolites. Metabolite production was expected to occur at a heightened rate in the 100% of 10 RM protocol as fast twitch muscle fibers, particularly the type IIb are being recruited. The results of this study revealed no difference between the protocols for lactate production. It may be that total lactate production within the forearm flexion exercise was not large enough, especially when the measurement used was not intra-muscular. Furthermore, the measurement occurred following the final set of forearm flexion, after both groups had incurred volitional muscle fatigue, promoting similar acute neuromuscular demands.

Maximum Isometric Voluntary Contractions

The results of the MVIC are consistent with the results of previous research examining isometric and dynamic muscle contractions. In the present study MVIC decreased on average by 18% in the 90% of 10RM protocol and by 19% in the 100% of 10RM protocol, with no significant difference between the protocols (Fig. 4). Behm, Reardon, Fitzgerald, and Drinkwater (2002) observed that MVIC was depressed by 18.1 to 21.4% after one set of a 5 RM, 10 RM or 20 RM elbow flexion exercise, demonstrating there was no difference in the magnitude of the decrease in MVIC between the three conditions. However, in the present study and Behm et al. (2002), the training protocols involved dynamic exercise, whereas the assessment of maximal force was isometric. The differences in contraction type between the fatiguing protocols and maximal strength testing may have misrepresented the magnitude of neuromuscular fatigue and could have accounted for the similar decrements in MVIC experienced by both protocols.

Linnamo, Hakkinen and Komi (1998), used a heavy strength training protocol that consisted of performing 5 sets of bilateral leg extensions using a 100% of 10 RM load. The male

subjects experienced a 24% reduction in MVIC following the heavy strength protocol. The 19% reduction in force incurred over 3 sets in the current study seems consistent with other muscle groups in similar subject populations.

However, the differences in volume observed cannot be explained from current MVIC results because both protocols produced similar decrements in force after the 3 sets of training. Given, that the amount of force a muscle can contribute is directly related to the maximal momentary force producing capabilities, it might be speculated that individuals in the 90% of 10 RM protocol had a larger reserve of force throughout the 3 sets. However, blood lactate results did not support this observation. No significant differences existed post exercise between the two protocols for blood lactate, suggesting that similar levels of fatigue had occurred.

Integrated Electromyography

The current results revealed a significant 17% and 18% decline in $iEMG_{max}$ in protocol A and B, respectively (Fig. 6), which are comparable to the findings of Linnamo et al. (1998) following 5 sets of 10 RM leg extension. Furthermore, Behm et al. (2002) found a 20.5% reduction in the iEMG of the biceps brachii following a single set of either 5, 10 or 20 RM. Although the decrements in $iEMG_{max}$ suggest a reduction in muscle activation, it was not possible to determine from the current protocol if this resulted from central or peripheral mechanisms (Linammo et al., 1998).

The results upon first analysis seem to indicate that changes in $iEMG_{max}$ closely reflect changes in MVIC. Mean percent differences from pre - to post - exercise (percentage difference pre values) revealed that, EMG incurred a similar drop-off in magnitude as MVIC. This might suggest that all changes in iEMG reflected neural mechanisms. However, this analysis is not

supported in Table 3, as no significant correlation existed between the iEMG and MVIC. Furthermore, these results are not consistent with research showing that EMG activity does not mimic changes in force observed with MVIC (Bigland-Ritchie et al., 1983). Closer examination of the results revealed similar percent mean decreases but different patterns of decline amongst the MVIC and $iEMG_{max}$; change in MVIC consistently decreased, but changes observed in iEMG were equivocal, signifying that several competing EMG responses or different individual responses, especially individual recruitment thresholds, may have resulted in increased neural drive in some individuals and a decrease in others. Given that the subjects in the current study were exercising on average at 76.2% of 1RM, this contention is consistent with the suggestions of Deluca et al. (1996), who suggested that the biceps or forearm flexor muscle group undergoes no further recruitment after 80% of 1 RM.

Similar trends existed for $iEMG_{mean}$ (Fig. 5). However, significant within protocol differences did not exist pre to post exercise. These results support the current findings, which suggest acute decrements in MVIC are not exclusively the result of changes in neural drive. It appears that peripheral or an interaction of peripheral and neural factors are responsible for changes in MVIC.

Changes in recruitment, firing frequency & synchronization have all been shown to contribute to the EMG signal (Behm, 1995). The possibility exists that under different fatigue conditions, the iEMG reflects the relative contribution of each neural mechanism (Green, 1990), sometimes increasing the signal (i.e. increased motor unit recruitment, synchronization, and firing frequency) sometimes decreasing the signal (i.e. asynchrony, decruitment, and decreased firing frequency). The response has been hypothesized to be dependent on the duration (Behm & St. Pierre, 1996) and intensity of the contraction (Hakkinen, 1994). Typically, increases in EMG

signal are observed during low to moderate intensity sustained contractions (Bigland-Ritchie, Furbush & Woods, 1986). Decreases in iEMG have been observed under conditions involving sustained maximum voluntary contractions (Hakkinen, 1994; Kent-Braun, 1999). Acute decreases in motor unit firing frequencies and reduced motor unit recruitment characterize alterations in central drive and may account for the decrements in muscle activation (Green, 1990).

Integrated EMG_{mean} showed no change pre to post for mean area under the curve. Mean area under the curve was measured .25 s after the onset of MVIC and under these conditions is said to be a much more stable measure of change in the electrical properties of muscle during fatigue (Deluca, 1997). The lack of differences from pre to post exercise, likely reflect the variability in individual response to the training protocol. It has previously been shown at tensions representing less than 80% of MVC there is a steady increase in the amplitude of the iEMG, indicating that active motor units are continuing to maintain force through increased firing frequency, synchronization or motor unit recruitment (Vitasallo & Komi, 1977). During maximal contractions (Bigland-Ritchie et al. 1986) or submaximal contractions carried to fatigue (Duchateau & Hainaut, 1993) a decrease occurs in EMG output over time. Given the use of submaximal intensities in the current protocols, increases or decreases in the iEMG could be expected. This is consistent with the current study, as no clear trend is apparent, leaving the possibility, that several opposing control strategies may have contributed to the iEMG signal on an individual basis. Some subjects decreased their $iEMG_{mean}$ response, whereas others increased their response from pre- to post- fatigue, but in all cases noticeable change occurred after 3 sets of forearm flexion.

Another possible explanation may be that both neuromuscular potentiation and fatigue co-exist during submaximal contractions (MacIntosh & Rassier, 2002). MacIntosh and Rassier argued that during a protocol involving submaximal intermittent isometric contractions a decreased activation (i.e. fatigue) of muscle is followed by eventual increases (i.e. potentiation). This response is likely individual, but potentiation has been found to be greatest after 15 s of maximal sustained contraction and thereafter decreases (Barry et al., 2000). Potentiation was still noted after 20 s of maximal isometric contraction. The inconsistent iEMG response between subjects might be explained by the existence of potentiation. Subjects in the current study performed 10 or less contractions lasting between 18 and 30 s, suggesting that potentiation could have either been masking the effect of fatigue.

Explanations for differential recruitment patterns do exist under conditions of imposed fatigue and below full motor unit recruitment threshold. One strategy to maintain force is to recruit available motor units and to continue to recruit new motor units over the 3 sets (Rooney et al., 1994). This recruitment strategy is utilized differently between individual muscles (Deluca et al., 1996) and is likely dependent on numerous factors, including previous training experience (Benardi et al., 1996), the muscle being tested (Kulkulka & Clamann, 1981), and the level of activation that a muscle can obtain (Belanger & McComas, 1981). Furthermore, recruitment of new motor units can occur through a larger range of RM efforts when training with a new task (Benardi et al., 1996), suggesting that the recruitment pattern of a muscle is dependent on the implied demands of the task. Unfamiliarity with a task is accompanied with increased neuromuscular demand. This is consistent with the concept that untrained individuals are unable to fully recruit all motor units during MVC (Belanger & McComas, 1981; Sale, 1988). Benardi et al. showed increases in median frequency (i.e. motor unit recruitment) occurred over 6 weeks

of training. Median frequency is a measure of motor unit recruitment. However, the task was novel and, although not stated, the subjects had no background in forearm flexion involving high forces, suggesting that less motor skill and a shorter practice period would be required of the experienced subjects in the current study.

This change in EMG would result in increased $iEMG_{max}$ and $iEMG_{mean}$. In the current study the possibility existed that some of the less experienced subjects, especially with this particular forearm movement, were training at a lower relative intensity than the more experienced lifters, who previously trained specifically in the elbow position required in the current study.

Another possibility for the increased iEMG activity in a fatigued muscle might involve fiber type availability. Type IIb muscle fibers require the highest intensities in order to be activated (Fleck & Kraemer, 1998), suggesting that in some subjects, particularly those inexperienced with the forearm flexion, the fibers with the greatest fatigability and contribution to iEMG signals were not available or required to maintain force during set 1 but were recruited during subsequent sets to maintain force as other fibers became fatigued. The results were consistent with this theory. Examination of the individual data revealed four of the five subjects who decreased their signals had the greatest training experience in various forms of forearm flexion.

Similarly, chronic resistance training with heavy load, will lead to the recruitment of previously unavailable fibers. Furthermore, the type of adaptation associated with high intensity resistance training is assumed to be primarily neural in nature (Sale, 1988). Given that full recruitment has been found to occur at approximately 80% of 1RM for the biceps brachii and forearm flexors (Deluca et al., 1996), and that experience may effect the availability of

previously unrecruited motor units, it has been suggested that less trained individuals train at a relative lower intensity than more experienced individuals (Wilson, 1995). On average the subjects in the current study were training at 73% and 76% of 1RM for protocols A and B, respectively, suggesting that the training intensity in the current study would have allowed initial increases in iEMG followed by decreases as decruitment occurred without further new recruitment. This would likely have occurred during sets 2 and 3, as 3 min has been shown to be an insufficient amount of recovery time with similar training intensity (Pincivero, 1998). If this holds true for forearm flexion, and the current drop-off in volume for protocol A supports this contention, then subjects performing Protocol A would have a greater fatigue induced state from the previous set prior to the beginning of the subsequent sets when compared to protocol (B). This could potentially place greater stress on the neuromuscular system and lead to increased neural drive for subjects performing protocol A. However, because some subjects increased neural drive across sets and other subjects decreased, it seems that the response to fatigue is individual and dependent on a number of factors.

Other neural mechanisms such as co-activation, which have been found to increase the coordination of a muscle, may contribute to increases in recruitment and the resultant neural drive (Rutherford & Jones, 1984; Behm, 1995). When prime movers are fully activated, increases in neural drive may be the result of input from synergistic or stabilizer muscles permitting optimization or maintenance of steady force output or decreases in antagonist activity (Behm, 1995). Similarly, Rutherford and Jones (1984) stated that a large part of the improvement in the ability to lift weights was due to an increased co-ordination of other muscle groups, such as the ones utilized in stabilizing or improving the precision of movement. In addition, Nakazawa et al. (1993) have shown that the brachioradialis becomes more active as the elbow

becomes more flexed, especially during eccentric and isometric contractions, similar to those required in our experimental design. Given, that this muscle lies posterior to the biceps brachii, it is possible that surface iEMG may be detecting activity from underlying muscles, such as the brachioradialis.

Approximately half of the subjects in the current study decreased their iEMG response. Under conditions in which force is no longer being maintained (i.e. the end of a set), an overall decrease in iEMG response might be expected. Several studies using maximum concentric intensity have found this response (Bigland-Ritchie, Johansson, Lipold & Woods, 1983; Bigland-Ritchie et al., 1986).

The results from the current study seem consistent with Benardi et al. (1996) who found that the control strategies (i.e. relative contributions of firing rate and fiber recruitment) employed are dependent on the type of contraction and the muscle being tested (Behm 1995; Kulkulka & Clamann, 1981). Decreases in iEMG are consistent with studies showing a decreased neural drive from either decreases in motor unit recruitment (Kulkulka & Clamann, 1981) and (or) decreased firing rates (Deluca et al., 1996).

Deluca et al. (1996) found that the firing rates of the tibialis anterior and first dorsal interosseus decreased over an 8-15 s isometric contraction. Furthermore, the decrease in firing frequency was not accompanied by an increase in motor unit recruitment, especially during contractions at 80% of MVC, suggesting that above motor unit range, modifications in force output are a result of rate coding. It might be speculated that distal muscles, those requiring fine motor control, may use recruitment primarily with lower intensities, with force modifications for higher intensity being the result of changes in firing frequency. Subjects in the current study were working at approximately 73 % and 76% of 1RM during the 100% and 90% of 10RM

protocols, respectively. It might be concluded from the current study that both control strategies were contributing to the iEMG signal, which in some cases led to increases in neural drive (i.e. increased firing frequency or motor unit recruitment) and in others decreased neural drive, which had an opposing effect on the iEMG signal. The variability in response collectively contributed to no change in the $iEMG_{mean}$.

Furthermore, Behm et al. (2002) observed no difference in muscle inactivation following three different fatiguing loads for forearm flexion. The level of inactivation was not different between protocols, although different levels of fatigue were noticed. These results suggest that fatigue is both neurally and peripherally mediated. Similarly, changes in iEMG have been correlated to changes in the peripheral measures of fatigue (Brandenburg et al. 2000; Kent-Braun, 1999). Support for a link between peripheral anaerobic metabolism and neural / central fatigue exist (Kent-Braun, 1999). Kent-Braun suggested that an association exists between ion production and EMG, and is related to a feedback mechanism associated with peripheral mechanisms. These mechanisms are said to affect descending motor drive during maximal efforts. MacIntosh and Rassier (2002) indicated that several competing mechanisms, suggesting fatigue and post activation potentiation are present at the same time. It is possible that independent peripheral mechanisms are responsible. Alternatively, central and peripheral mechanisms may be simultaneous and opposite in nature. Fatigue and potentiation seem to occur simultaneously during intermittent, incompletely fused tetanic contractions (MacIntosh & Rassier). MacIntosh and Rassier reported that fatigue was evident after just 5 stimulating pulses and it was suggested that metabolic or peripheral limitations were not involved in the response. Based on these studies, it might be speculated that a feedback system exists in the periphery to

Furthermore, it might be expected that the 100% of 10 RM would have accumulated larger increases in blood lactate. As both groups incurred volitional fatigue during the final set it might be concluded that acute fatigue of set 3 masked the cumulative fatigue. Furthermore, given that lactate measures were taken pre and post exercise it was impossible to observe changes between set 1 and 2 or set 2 to 3.

Past research has indicated that decrements in force output are related to changes in pH and metabolic by-products such as lactic acid (Bogdanis et al., 1996; MacDougall et al., 1999; Tesch et al., 1983). Theoretically, preventing the release of Ca^{2+} at the sarcoplasmic reticulum (Favero, Zable, Colter, Abrasom, 1997) leads to a reduction of the ability of troponin to expose attachment sites on tropomyosin. Without Troponin exposing the binding sites, myosin binding is limited, resulting in a reduction in force development. Through feedback to supraspinal pathways, neural drive may be decreased causing the shape and frequency of action potentials to be altered. These changes could therefore lead to a decrease in the EMG signal. Given that changes in pH and blood lactate are distal to the neuromuscular junction (McLester Jr., 1997), the mechanism involved is considered peripheral rather than central. The current results do not support this contention. No significant association between blood lactates and MVIC was observed for either protocol (Table 2.). This may be explained in part because recorded blood lactate levels were low.

Previous research has shown a link between blood lactate and strength development (Rooney et al., 1994) and muscle hypertrophy (Schott, McCully & Rutherford, 1995). Typical strength and hypertrophy programs in the past have typically involved training to volitional failure on every set. One characteristic of training to failure is "the burn", related to high levels of lactic acid. Increased lactic acid production leads to increased acidity within the muscle. If

lactic acid is related to muscle adaptation then training techniques producing the highest levels would represent the optimal stimulus. Since the protocols used in the current study produced no difference in blood lactate response at the end of three sets, it can be assumed that both protocols are equally effective in contributing to muscle adaptation. However, as testing only occurred prior to and after completion all 3 sets of resistance training and both protocols involved a temporary state of fatigue at the end of third set, it is not possible to determine how acute changes in blood lactate differed during sets 1 and 2. Furthermore, this design prevented any conclusion relating acute changes to chronic adaptation.

Chronic adaptation from training to failure may involve an increased ability of involved muscles to buffer lactic acid, limiting the negative impact on force production, compared to those individuals that train with powerlifting methods (Tesch, Thorsson, & Kaiser, 1984). This observation seems to be supported by the current results. Several of the subjects in the current study, especially those with endurance training backgrounds were able to maintain near maximal efforts (i.e. no drop-off) throughout the 3 sets of 10 RM training. In addition those subjects also showed the smallest post exercise blood lactate response. Similarly, the individuals with the highest drop-off in volume had training backgrounds representative of powerlifters. It is likely that lactate or metabolites related to lactate production are filtered at a higher rate due to adaptations associated with this type of training, permitting a quicker recovery between sets. How lactate accumulation contributes to myogenic adaptation is unclear.

It is assumed that increases in blood lactate concentration reflect an accumulation of muscle lactate and a resultant decrease in muscle pH (Tesch, Collainder, & Kaiser 1986; Abdessemed et al., 1999). Research has indicated that reductions in muscle pH and not increases in lactate inhibit the function of the contractile proteins, consequently impairing the maximum

amount of force that can be generated (Kent-Braun, 1999; Linnamo et al., 1998). In humans a temporal relationship has been found between the reduction in force and pH level (Cady, Elshave, Jones, & Moll, 1989). However, Beliveau, Helal, Gaillard, Van Hoecke, Atlan, & Bouissou, (1991) and Miller, Boska, Moussavi & Weiner (1988) found that EMG signals were fully recovered although blood lactates had not reached peak values, making it difficult to assume a causal relationship between blood lactate and force reduction in skeletal muscle. The current research suggests that changes in lactate are not related to the decreases in maximal force. This result seems consistent with previous research examining the contribution of lactic acid to a decrease in force (Tesch et al., 1983; Fitts & Balog, 1996; Linnamo et al., 1998; Kent-Braun, 1999) or force producing capabilities of the muscle (Favero et al., 1997). Recent evidence has indicated acidification has little direct effect on force production, especially during the early phase of muscle contraction (Westerblad et al., 1998). It was suggested that three distinct phases, involving different metabolites exists. Westerbald speculated that rate of cycling cross bridges is little affected by acidosis at physiological temperatures and that the inability of cross bridges to produce energy is more likely related to changes in inorganic phosphate (Pi). However, this research involved individual skinned muscle fibers.

It has been suggested that muscle fatigue, especially the fast recovery phase (Westerbald & Allen, 1996), also results from the depletion of PCr stores (Abdessemed et al., 1999; Bogdanis et al., 1996; Rooney et al., 1994; Tesch et al., 1986). Specifically, declining PCr stores reduces the availability of Pi for ATP resynthesis, identifying a potential role of Pi in reduced force production. However, phosphocreatine is known to be a short term energy source which is exhausted relatively quickly (i.e. < 10 s). Because, the current protocol would have required energy supply from several metabolic pathways, and no correlation existed between blood lactate

and the change in force or volume, it might be speculated that other metabolic by-products like P*i* are being produced simultaneously. Understanding fatigue may require a more comprehensive examination and understanding of metabolic interaction than the current protocol permitted.

It is well known that PCr stores are replenished in a short period of time (i.e. < 1 min) and by 3 min are at near maximal levels (McCardle et al., 1996). These results are further supported by research from our lab (Bradenburg et al., 2000), in which subjects performed 6 sets of 6 RM training to failure. Almost all subjects recorded a significant drop-off in the number of repetitions performed over the session but were able to perform the first 3 repetitions during forearm flexion for each set. Bogdanis et al. (1996) examined this phenomenon using cycle ergometer sprint training with 4 min of rest between 30 s bouts of maximal effort at high intensities. Results indicated, similar to Abdessmed et al. (1999), that there was a correlation between the recovery of power output and the recovery of PCr during the first 10 s of pedaling. The measure of PCr restoration was taken at 3.8 min of recovery. Furthermore, although pH remained unchanged during the 4 min of recovery, PCr levels were restored to 78.7% of their resting value. Cumulatively, the research seems to indicate that several mechanisms are involved in the fatigue process, suggesting that the phoshocreatine (PCr) system and lactic anaerobic metabolic pathways have independent effects on high intensity muscle fatigue.

Summary

The present study supports the conclusions of Vestergaard-Poulson et al. (1993) and the assumptions of Baker (1998). Stopping contractions short of volitional failure may prevent large metabolic shifts, permitting quicker recovery between sets. If this limit is exceeded, as found by

Vestergaard-Poulson et al., it appears that recovery from exercise is prolonged and prevents further force production for greater than 30 min.

It is well known that the structure of a resistance training program influences the type of muscular adaptation. Less is known of the acute physiological responses that affect these differential responses. It might be that both changes are occurring simultaneously, preventing distinction regarding the effects of either one.

While protocols used during this study were quite similar in intensity, the volume performed during each protocol was different. Based on these observations, the current results do not support research showing that the structure of a resistance training protocol influences the amount or site of accumulated fatigue (Bosco, Colli, Bonomi, Von Duvillard & Viru, 2000). Changes in MVIC, iEMG, and blood lactate were similar, regardless of the intensity used. If the site of fatigue differs depending on the structure of the prescribed resistance training, it might be speculated that the specific neuromuscular responses may depend on the degree of acute neuromuscular fatigue induced by the prescribed protocol. Our results may be a consequence of the similar intensities used (90% of 10RM Vs 100% of 10RM). Previous research has found that at intensities beyond 80% of 1RM no further recruitment of motor units occurred (Deluca et al., 1996). Upon completion of the third set, it might be speculated that the subjects were working at an intensity equal to 80% of their current 1RM. This is supported by Fleck and Kraemer (1998) who suggested that on average most subjects perform approximately 10 repetitions when using 80% of their 1RM. Further, the 90% of 10RM group completed a higher volume of work, which should have contributed to changes in the measured variables, preventing conclusions regarding the effects of the structure of resistance training programs on acute and chronic neuromuscular responses. Previous research does not support our findings of similar neuromuscular responses

to resistance training (Bosco et al., 2000). Other training variables, including intensity (Brandenburg et al. 2000; Tan, 1999), experience (Linnamo, Hakkinen, & Komi, 1998), rest interval length (Pincivero, et al., 1997; 1998; Abdessemed et al., 1999) and exercise duration (Behm & St. Pierre, 1994; Behm et al., 2002) may all influence the magnitude and perhaps the site of neuromuscular fatigue.

Furthermore, the different fatigue sites (central and peripheral) involve fatigue mechanisms which have distinct temporal characteristics (Fitts & Balog, 1996; Westerblad et al., 1998), suggesting that the resistance training prescription may influence the site of fatigue. Electromyography studies have shown that the maximum iEMG signal recovers at an accelerated rate compared to muscle metabolites (Miller et al., 1988), suggesting that the central nervous system is capable of intiating force, but alternative sites, distal to the neuromuscular junction are preventing maximal isometric contraction from occurring.

If fatigue is controlled by several underlying mechanisms, which may be differentiated based on the exercise intensity (Behm, 1995; Westerblad et al., 1998; Brandenburg et al., 2000), duration (Behm & St Pierre, 1994; Westerblad & Allen, 1996), and rate and time of contraction (Fitts & Balog, 1996), then the neuromuscular effects of maximal strength resistance training protocols (> 3 min rest) may be different from hypertrophy protocols (< 3 min rest) that are typically used.

Although studies on acute design do not establish a causative relationship it might be speculated, given the probable role of volume as a stimulus for muscular adaptation (Baker et al., 1994; Kramer, Stone, O'Bryant et al., 1997), that maximizing this resistance training variable through the manipulation of fatigue could be beneficial in program design. In accordance with EMG studies there appears to be a rapid return to force producing capabilities of a muscle as long

as a critical point of metabolite production is not exceeded. In "Fatigue Experiments" electromyographic activity has been completely recovered prior to blood lactate or muscle pH (Beliveau et al., 1991). If three distinct phases of fatigue exist, and if fatigue is indeed a stimulus for strength (Rooney et al., 1994) and hypertrophy (Baker et al., 1994, Schmidbleicher & Buerrhle, 1987), prescription of strength training programs should take into account all phases of fatigue. Baker (1998) speculated that 90% of individuals, 10 RM utilized when programming 3 sets of resistance exercise. Potentiation studies have shown that performance of isometric contractions lasting 15 s, after which the ongoing process of fatigue becomes dominant (Barry et al., 2000), reduces the force producing capabilities of a muscle. This phase of fatigue is likely due to increased metabolites (i.e. lactic acid and Pi) and reduced release of Ca^2 from the sarcoplasmic reticulum (Westerbald et al., 1998; Favero et al., 1997).

Increased myogenic stress may also result in increased myofibrilar damage, resulting in myofibrilar adaptation (i.e. hypertrophy). Furthermore, from a motor learning perspective, precluding early fatigue may enhance motor skill development by increasing the likelihood of promoting a memory engram (Thompson, 2000), which is a distinct memory location, that is dependent on consistency of movement. Therefore, an increased efficiency in the ability to learn a motor skill may occur. Following the recommendations of Baker, preventing early fatigue could theoretically permit potentiated neuromuscular drive to a muscle prior to competition (or training). Through the delay of fatigue, practitioners, through careful planning, may be able to optimize several important aspects of training. Based on the subjects in the current study individuals could be expected to perform all 10 repetitions on consecutive sets at 90% of 10RM and for all three sets. Kraemer and Fleck (1998) have suggested for certain exercises that individuals can work at a higher relative percentage of their 1RM for a specific number of

repetitions. Programming for multi joint muscle groups may require some adjustment towards higher intensities. In general the 90% estimation can be used as a beginning point in exercise prescription with adjustments made according to the individual and type of lift performed.

Further scientific analysis should involve long term training programs and specific dose-response relationships for each muscle group. Determining how the ongoing processes of fatigue and potentiation interact may also help researchers and practitioners develop appropriate programming for specific neuromuscular adaptations. Changing the level of fatigue may help optimize neuromuscular adaptation but other factors known to be related to neuromuscular adaptation will differentially affect the results of any long-term training program.

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Appendix A: Limitations, Assumptions and Definitions

Limitations/ Delimitations

This study examined a group of well trained, college-aged, male subjects.

All subjects had similar but not identical levels of absolute strength . This was important because the absolute load lifted has been shown to increase the level of exercise induced lactic release (Hickson, Hidaka, Foster, Falduto, & Chatterton, 1994).

Acute blood Lactate production has not been linked to physiologic adaptation, preventing the conclusion that an increased or reduced acute lactate response affects the chronic adaptive process.

It is likely that iEMG cross talk did occur, and it is impossible to know if the prime mover was responsible for the entire signal input?

Assumptions

It was assumed that all participants had rested for 48 hours from all resistance exercise involving the particular muscle being tested. It was assumed that all efforts during the RM testing were maximal, and that lactic acid produced in the biceps was accurately reflected from blood samples are taken from the finger. It was assumed that the Modified Borg Scale was representative of lactic acid accumulation in resistance exercise.

Operational Definitions

Dynamic muscle action: A muscle action in which the external resistance or load does not change and that both a lifting and lowering phase occur during each repetition (Fleck & Kraemer, 1998).

Dynamic constant external resistance (DCER): A form of resistance training involving a dynamic muscle action (i.e. the load or resistance does not change during the concentric or eccentric phase of each repetition) (Fleck & Kraemer, 1998).

Isometric muscle action: A muscle action in which a muscle is activated and develops force but no movement at a joint occurs (Fleck and Kraemer, 1998).

Failure: Temporary volitional fatigue at the end of the tenth repetition (i.e. the end result of reduced force production capabilities of the active muscle(s) to a point where the muscles are no longer able to perform another repetition at a 10 RM resistance).

Lactic acid: A needed source of chemical potential energy continually utilized by the body during moderate exercise and accumulates during intense/heavy exercise (McCardle, Katch & Katch, 1996).

Relative intensity: The percent of 1 repetition maximum (1RM) which is used as a training load (Stone ,1998).

Repetition: The number of work intervals in 1 set. For the purpose of this study it was defined as the number of consecutive work intervals involved in both eccentric and concentric muscle actions (Bompa, 1996).

10 RM: The maximum load that can lifted for a set number of repetitions, (Bompa, 1996), thus 10 RM was defined as the maximum load that a muscle or group of muscles could lift for 10 repetitions before failure occurs.

Resistance training: The use of various methods or equipment to provide an external force against which to exercise (Bompa, 1996).

Rest interval (R.I.): The time from the completion of one set to the onset of the next.

Set: A combination of repetitions performed to failure, or to a specified number, and performed consecutively.

Strength: The maximal amount of force that a muscle or muscle group can exert in a specified movement pattern at a specified velocity of movement (Knuttgen & Kraemer, 1987). 1RM is typically used as a measure of strength.

Volume: The amount of work performed per repetition, per set, or per workout (Stone, 1999). Total work per set was defined by Volume Load (i.e. sets x reps x load).

Appendix B: Review of Literature

Introduction

Fatigue during resistance training has been described as a decrease in the force producing capabilities of a muscle (Kent-Braun, 1999) or an eventual inability to produce a desired force (Enoka & Stuart, 1992). It has been related to the intensity or the percentage of 1 RM that is being used during a training session (MacDougall, Sale, McCartney, Lee, & Garner, 1999) such that an individual using 80% of 1 RM, reaches failure during a given set when the force producing capabilities of the muscles involved become reduced by 20%. 'Failure' has been described as temporary volitional fatigue against a set load (Stone, Chandler, Conley, Kramer & Stone, 1996) or the inability to complete 1 more repetition at the resistance being used. The terms failure and fatigue are often used interchangeably. For the purpose of this review, fatigue during resistance training will be an ongoing decrease in performance resulting in a state of temporary muscle failure.

Consecutive sub-maximal contractions will eventually result in temporary volitional fatigue. However, there are complications that affect the study of fatigue. Several mechanisms have been proposed to contribute to decrements in performance, but less is known regarding the relative contributions of these mechanisms to fatigue is not well understood. .Further confounding the study of fatigue is research suggesting that both neuromuscular potentiation and fatigue co-exist during involuntary muscle maximal stimulation (MacIntosh & Rassier, 2002). Unusual fatigue responses resulting first in decreased (i.e. fatigue) then increased (i.e. potentiation) of force have been noted

(MacIntosh & Rassier, 2002). This observed reversal of fatigue response complicates the interpretation of previous results examining fatiguing contractions.

Central mechanisms are those that effect (i.e. increase or decrease) neural drive and are proximal to the neuromuscular junction (NMJ) (Kawakami, Amemiya, Kanehisa, Ikegawa, & Fukunaga 2000) while peripheral factors are considered metabolic (Fitts, 1994) or structural in nature (Green, 1997) and are distal to the NMJ (McLester Jr., 1997). It has become widely accepted that peripheral fatigue is the major contributor to inability to maintain the initial maximal force. However, it has suggested that experimental observations should be examined more carefully in order to determine if 'proximal' changes in force cause changes in force production or merely accompany fatigue (Gandevia, 2001).

Furthermore, it has been proposed that changing the protocols of a resistance training program will produce different acute and chronic physiologic responses (Tan, 1999; Kraemer, Duncan & Volek, 1998; Fleck & Kraemer, 1998). Most research involving strength training and program prescription requires subjects to perform repeated lifts without resting until the subjects are unable to voluntarily execute further lifts (i.e., temporary volitional fatigue/failure). Such protocols are based on the assumption that muscle failure is required to produce full motor unit activation (MUA). However, there is limited experimental evidence to support the need for muscle failure and theories of motor unit recruitment. Some studies have found that suggesting that motor units are fully recruited at a lower repetition maximum (i.e. % of 1 RM) than once believed (Behm, 1995; Kulkulka & Claman, 1983, Deluca, Foley & Erim, 1996; Tesch, Ploutz-Snyder, Ystrom, Castro & Dudley, 1998).

It has been suggested that volitional fatigue was necessary in order to recruit and derecruit the maximal number of available fibers. Several reviews have examined the practice of repetitions to failure during dynamic resistance training (Baker, 1998; Kraemer et al., 1998; Stone et al., 1996); it has been suggested that training variables such as volume and intensity, may be more or equally as important as muscle failure at eliciting a training response, especially as they pertain to strength and hypertrophy adaptations. Baker (1998) stated that training involving volitional fatigue with inadequate rest prevents individuals from optimizing the total volume of work performed during a resistance training session. It might be suggested that resistance training which optimizes total volume, inter-set rest intervals and the intensity of exercise, while promoting volitional muscular fatigue may produce the best gains in strength and hypertrophy.

Although the processes associated with fatigue have been empirically examined for some time, scientific understanding of how training variables related to fatigue (i.e. volume, intensity, inter-set rest interval and temporary volitional fatigue) effect acute and chronic neuromuscular adaptations is lacking. Therefore, purpose of this review will be to examine physiological mechanisms associated with both peripheral and central fatigue, as well as how manipulations of training variables during resistance training programs can optimize the acute physiological responses which may help promote chronic muscle adaptation

Maximal Voluntary Contractions

Research has shown that force is significantly reduced with multiple sets of intense training in men (Behm, Reardon, Fitzgerald & Drinkwater, 2002; Brandenburg, Docherty & Benson. 2000; Hakkinen, 1992; Hakkinen & Linnamo, 1998) and women (Hakkinen, 1993). Decreases in maximal force measurements as the result of submaximal resistance training protocols have been associated with peripheral and central mechanisms. During fatigue protocols, force measurements usually involve maximum voluntary isometric contractions (MVIC) prior to and after performing the fatiguing protocol. Observations of force reduction over repetitive sets of submaximal resistance training has also been related to a 'drop-off' in the desired number of repetitions on sets subsequent to the first set that was performed (Brandenburg, Docherty & Benson 2002; Macdougall et al. 1999).

Observations indicate that the magnitude of MVIC reduction is influenced by the intensity of the resistance training protocol (MacDougall et al. 1999). However, Behm et al. (2002) observed that MVIC was depressed by 18.1 to 21.4% after one set of a 5 RM, 10 RM or 20 RM elbow flexion exercise, demonstrating there was no significant difference in the magnitude of the decrease in MVIC although different training intensities were used during the 5 RM, 10 RM and 20 RM conditions. However, the training protocols used by Behm et al. involved dynamic exercise but the assessment of maximal force was isometric. Linnamo, Hakkinen and Komi (1998) observed similar decrements (23.7%) in force following 5 sets of 10 RM during leg extension exercise.

Hakkinen and Pakarinen (1993) observed a 10.3% reduction in force over 20 sets of 1 RM and a 24.6% reduction in force over 10 sets of 10 repetitions. However, these

researchers reduced the load prior to each set in order to maintain performance of the same number of repetitions on each set. Furthermore, comparison to the previous studies is difficult, as the reduction in force reflected changes in force after a 3 min rest interval instead of the force decrement being measured directly after completion of a set, which is typical of the designs for the previous studies. The results of these studies suggests that force decrement of muscle does not match the expected performance if the expectations are solely based on the percentage of 1 RM. This may represent a defense mechanism in muscle, whereby acute physiological responses (i.e. increased lactic acid or Pi production or lowered pH) prevent further decrease in essential energy sources, preventing levels of intramuscular ATP and Ca^{++} from reaching levels that would lead muscle distress.

Additionally, decrements in MVIC and the combined iEMG seem to be sensitive to the volume demands placed on the exercising muscle. Hakkinen (1994) examined neuromuscular fatigue of the quadriceps in male and female strength athletes following the performance of 10 sets of squats using a 10 RM load. Male strength athletes experienced a 47% reduction in MVIC with a corresponding 44% reduction in iEMG. Females revealed approximately a 32% decrease in MVIC with concomitant 29% decrease in iEMG. The larger reductions in MVIC and iEMG observed in this study, compared to the above-mentioned studies, may be attributed to a larger training volume or increased myofibril disruption that this type of loading protocol might be expected to cause.

Similarly, acute changes in maximal isometric force and iEMG were compared in response to a heavy strength training protocol and an explosive strength training protocol (Linnamo, Hakkinen & Komi, 1998). The heavy strength training protocol consisted of

performing 5 sets of 2 legged extensions using a 10 RM load. The explosive power protocol included performing 5 sets at a resistance representing 40% of 10 RM. Males experienced a 24% reduction in MVIC following the heavy strength protocol, whereas decreases in MVIC following the explosive training protocol totalled 11%. Female subjects demonstrated an 18.8% and 12% reduction following the heavy and explosive loading protocols, respectively. The reduction in MVIC following the explosive protocol was significantly less than that of the previously mentioned studies. However, during the explosive protocol the volume and intensity of training was significantly less and the rate of force development greater than the maximal strength protocols previously discussed.

Changes in MVIC represent neuromuscular performance decrements, as demonstrated through decreases in power output per repetition and drop off in number of repetitions. However, the exact site and mechanisms of performance decrement remain uncertain (Green, 1997). Researchers believe that as long as subjects are sufficiently motivated, peripheral mechanisms, including the depletion of muscle substrates, ionic imbalances, increased muscle acidity and alterations to contractile proteins are mainly responsible for acute neuromuscular fatigue (Behm, 1995; Bigland-Ritchie, Johansson, Lippold & Woods, 1983). Regardless of whether the source or site of fatigue is central or peripheral, decreases in performance associated with fatigue will always represent itself peripherally through interference with cross-bridge formation (Gandevia, 2001).

Brandenburg et al. (2000) examined the acute neuromuscular effects of subjects performing repetitions until muscle failure during protocols involving 6 RM and 10 RM. Despite the use of different intensities, decrements in iEMG & MVIC were similar. These results might suggest, similar to Deluca (1997) that decreases in the iEMG signal

associated with moderate intensity resistance training reflect changes in the force producing capabilities of the muscle. However, in order for this relationship to exist, the muscle under study must be fully activated (i.e. full motor unit recruitment, maximal firing frequencies etc), such that only decreases in performance are occurring. Furthermore, given that force decreases as a result of mechanisms that are both proximal and distal to the NMJ, integrated EMG response to moderate and high intensity loads might be influenced by a combination of both metabolic and neural fatigue. These results are not consistent with research demonstrating that EMG activity does not closely mimic changes in force observed with MVIC (Bigland-Ritchie et al., 1983). However, studies that have reported increases in iEMG after fatigue involve protocols of longer duration and lower intensities, preventing direct comparisons to moderate and high intensity protocols.

Mechanisms of Fatigue

Incomplete recovery during resistance training that can occur over several days is directly related to acute fatigue. The accrued fatigue from an acute bout of resistance training has been shown to last for as little as 30 s or as long as two weeks (Green, 1997). Therefore, it is likely that observable acute fatigue from resistance training results from both central and peripheral mechanisms (Kirkendall, 1990). This argument is supported by observations involving iEMG measurements. Integrated EMG has been found to return to pre-exercise values at a much faster rate than metabolic measurements during fatiguing (Miller, Boska, Moussavi, & Weiner., 1988; Beliveau, Van Hoecke, Garapon-Bar et al.,1992). This recovery may be affected by the type and intensity of the

contraction performed, such that 40 maximal eccentric contractions, performed for 3.5 s resulted in reduced iEMG values even after two days of recovery (Komi & Viitasalo, 1977). Such observations suggests that the mechanisms of fatigue are both metabolic and non-metabolic in nature (Green, 1997) and have been proposed to occur for several reasons, including failure of excitation coupling (Kirkendall, 1990) or structural damage, especially during eccentric contractions (Clarkson & Sayars, 1999).

Central fatigue is the result of poor motivation, increased inhibition of the motor neuron or recruitment. Acute increases and decreases in neural drive are most commonly associated with increased or decreased motor unit recruitment and/or motor unit firing frequency. To determine the amount of neural activation or inactivation researchers have most commonly used electromyography (EMG). An increase in the amplitude of the EMG signal is a result of greater muscle activation, while decreases in EMG amplitude is suggestive of less muscle activation. Both increases and decreases are considered to be the result of implied demands on the muscle being tested. In order to determine the effects of neural fatigue during a resistance training protocol, EMG is measured prior to upon completion of an acute bout. Furthermore, when the EMG signal is integrated (iEMG) the amplitude of the signal is related to increases or decreases in the amount of force produced by the muscle of interest (Deluca, 1997). Electromyography studies have been performed to examine neural effects on both maximal isometric muscle contractions (Bigland-Ritchie & Woods, 1984) and dynamic muscle contractions (Behm et al., 2002).

However, it may be that EMG readings reflect both central and peripheral fatigue and therefore may not be an effective measure of central fatigue. Gandevia (2001) has suggested that a reduction in the action potential derived from an active muscle fiber can

be the result of decreased descending drive from cortical pathways (i.e central) or a change in membrane potential / sarcolemmal function, which are considered distal to the neuromuscular junction and therefore peripheral in nature.

Neural Responses Involved in Fatigue

Recent research has shown that neural drive is significantly reduced with multiple sets of intense training in men (Hakkinen, 1992) and woman (Hakkinen, 1993). Behm et al. (2002) found a 20.5% reduction in the iEMG of the biceps brachii following a single set of either 5, 10 or 20 RM. Futhermore, Hakkinen (1992) examined acute neuromuscular responses to two successive strength sessions in the same day. The strength sessions involved different intensities, such that during the morning session subjects utilized 70-80% 1 RM, while the afternoon session involved sets ranging from 70-100% 1 RM. Researchers observed no decrement in EMG readings during or after the morning session the workout, but a significant positive correlation existed for the post-exercise measurements involving maximal force and iEMG readings after the second (afternoon) session. However, the lower intensity session always preceeded the higher intensity session, preventing the researchers from ruling out the residual effects of the morning session as a cause of decreased force during the afternoon session.

Assuming, that acute fatigue associated resistance training protocols begins to dissipate immediately upon removal of the resistance, it is important that post exercise measurements occur as quickly as possible after the completion of the last repetition. One technique used effectively in this manner is the interpolated twitch technique (ITT). The interpolated twitch technique involves stimulating the motor nerve of a muscle while

the activated muscle is simultaneously performing maximum voluntary contraction (Behm & St. Pierre, 1998; Kent-Braun, 1999; Gandevia, 2001). This stimulation occurs upon the cessation of the fatiguing protocol. If the stimulation causes an increase in the amplitude of the EMG signal above the maximal value elicited during MVIC, it suggests that neural factors have produced the observed decreases in performance.

Behm et al. (2002) utilized iEMG and interpolated twitch to determine acute neuromuscular responses after performing one set of 5, 10 or 20 RM loading protocols. Results indicated increased muscle inactivation through increases in interpolated twitch and decreases in iEMG signals for all three loading protocols. However, although the exercise intensities for each protocol were different, no differences existed in the extent of muscle inactivation between these protocols. From these results it would be concluded that significant neural fatigue resulted from an acute bout of exercise involving moderate to heavy resistances. However, the differences in contraction type between the fatiguing protocols and maximal strength testing may have misrepresented the magnitude of neuromuscular fatigue and could have accounted for the similar decrements in MVIC experienced by both protocols.

Other research has suggested that central fatigue has a negligible impact on acute performance as long as subjects are sufficiently motivated (Bigland-Ritchie et al., 1983; Behm, 1995; Gandevia, 2001). This observation is supported by research examining changes in iEMG in combination involving a normal mass action potential (M-wave). Bigland-Ritchie and Woods (1984) found no observable reductions in the M-wave when a muscle or muscle nerve is directly stimulated after an acute bout of fatiguing contractions. However, Behm and St-Pierre (1997) observed that measurements of M-

wave activity are dependent on the protocol used. During a short duration (4 min, 17 s) fatigue protocol involving repetitions performed at 50 percent of 1 RM, M-wave activity of the quadriceps was potentiated (16.7%), but was depressed (15.7%) when the measurement involved the plantar flexion. Because the extent of muscle activation and M-wave muscle activity contribute to iEMG signal, it is difficult to conclude from observations that the iEMG signal is not affected by neural mechanisms. Difficulties in measuring neural fatigue are supported by Barry, Sleivert and MacIntosh (2000) who results indicated that both increases (i.e. potentiation) and decreases (fatigue) in neural activity might to be simultaneous, yet competing responses during short duration moderate to high intensity resistance training protocols. Short duration intense muscle contractions lead to an early increase in muscle activation that is quickly overcome by the level of accrued fatigue, which is theorized to begin at the onset of moderate to intense resistance training (Gandevia, 2001; MacIntosh & Rassier, 2002). Therefore, decrements in muscle activation, could lead to a decreased iEMG in absence of any changes in M-wave, especially if both neuromuscular fatigue and potentiation are acting equally but oppositely. Behm and St-Pierre observed a 14.1% decrease in muscle activation (i.e. peak twitch torque) in conjunction with potentiation of the quadriceps m-wave, lending support to Barry et al. Furthermore, observations indicate that the EMG response may be specific to the motor unit activation patterns of individual muscles (Gandevia, 2001).

Further complicating observations of neuromuscular fatigue through iEMG are suggestions that iEMG signals are effected through peripheral mechanisms (Brandenburg et al. 2000; Kawakami et al., 2000, Gandevia, 2001). _Kawakami et al. Suggests the

possibility of feedback between the two systems based on observations revealing of an association between blood lactate and iEMG.

Peripheral Fatigue

Muscle Substrates

When sufficient rest for complete recuperation is not given maximum force generation cannot be maintained (Brandenburg et al., 2000; MacDougall, et al., 1999; Pincivero, Lephart & Karunakara, 1997). Ultimately, this means the supply of energy through ATP hydrolysis is less than the demand of ATP for continued performance of muscular contractions. During moderate to intense muscular contractions it appears that muscle glycogenolysis and and PCr breakdown are the primary sources of ATP re-synthesis (MacDougall et al., 1999). Further, it has also been shown that intramuscular stores of creatine phosphate and glycogen can adversely affect performance of submaximal muscle contractions (Green, 1997; MacDougall et al., 1999). Blood lactate is an arterial measure of lactic acid, which is a needed source of chemical potential energy, continually utilized by the body during moderate exercise but accumulates during intense/heavy exercise (McCardle, Katch & Katch, 1996). Therefore, studies examining the effects of fatiguing resistance training protocols on blood lactate concentrations have been performed in order to determine resistance training regimens that promote the greatest measurable levels of fatigue. However, very little research has examined the rate of supply of these substrates during moderate to high intensity activity.

Research on the role of blood lactate in performance of moderate to high intensity resistance training has been equivocal. Abernathy and Weir (1998) examined lactate

concentrations after 3 sets of 5 RM and 15 RM leg press in recreationally trained subjects. The rest between sets was 6 min, yet increases in blood lactate still occurred from set 1 – 3 during both protocols. However, although not clearly indicated in the results, there was no indication of reductions in volume over the 3 sets, suggesting that subjects were able to maintain performance despite increases in blood lactate. These results suggests that the time for complete recovery of performance during resistance training is much quicker (i.e. 3 – 5 min) than the time required for complete blood lactate recovery, especially during low volume, high intensity protocols. Therefore, blood lactate may be a poor indicator of muscle recovery following high intensity resistance training protocols.

Blood lactate is produced as a result of glycogenolysis (McCardle et al. 1996) and has been associated with decreases in pH (Cady, Elshove, Jones & Moll, 1989; McCartney, Spriet, Heigenhauser, Kowalchuk, Sutton & Jones, 1986). However, during intermittent submaximal muscular contraction lasting less than 10 s, it might be speculated that that decreases in performance are related to a reduction in creatine phosphate. In contrast, research has indicated that when inter-set rest intervals are greater than 1 min the reduced performance is the result of a decreased rate of glycogenolysis (i.e. inability to breakdown stored glycogen at a fast enough rate). This observation is supported by Green (1997) who proposed that creatine phosphate system may re-supply ATP at a rate far exceeding the utilization of ATP during high intensity exercise. While it would seem logical to supply ATP through the hydrolysis of CP it is suspected that the CP re-supply of phosphates is limited as a defense response preventing ATP from being exhausted to the point where autonomic functions would be affected (Green, 1997).

Furthermore, experimental evidence has found that CP is 90% resupplied after 1 min (McCartney et al., 1986). Perhaps, humans have a much larger store of glycogen to ensure preferential utilization of the slower supply of energy to prevent depletion of ATP stores. Therefore, it might be reasonable to assume that the rate of glycogenolysis is the limiting step in ATP supply during moderate to high intensity submaximal muscular contractions.

This hypothesis is supported by Robergs, Pearson, Costill, Fink, Pascoe, Benedict, Lambert, & Zachweija, (1991) and Tesch, Ploutz-Snyder, Ystrom, Castro, & Dudley, (1998) have examined fiber specific glycogen depletion utilizing protocols involving different exercise intensities and found that the type IIa and type IIb fibers are activated at much lower levels of tension than previously thought. Roberg et al. examined glycogen depletion in slow and fast fibers for knee extensions performed with a load equal to 35 percent or 70 percent of the 1 RM. Total work was partially equated between regimens because twice as many repetitions were performed with the lower load. Fast fibers showed greater glycogen loss than slow fibers for both loads. The greater glycogen loss may reflect greater glycolytic capacity within fast compared to slow twitch fibers.

Tesch, Ploutz-Snyder, Ystrom, Castro & Dudley (1998) expanded upon these findings by examining mixed muscle and single fiber glycogen loss during low to moderate intensity resistance exercise. Observations revealed a loss of glycogen in type IIa and IIb fibers, which was dependent on the exercise load used. With light loads (i.e 30% 1-RM) type IIa and IIb fibers showed no appreciable glycogen loss, but with loads representing 60% of 1 RM, significant glycogen depletion occurred in both type IIa and IIb fibers. However, order effects cannot be ruled out because the testing protocol

always involved subjects receiving the 30% load prior to the 60% load. It could be argued that the first of the two bouts of resistance training resulted in significant glycogen loss in type IIa fibers. Without proper recovery, type IIb fibers might have been preferentially recruited during set 2. Given, preferential loss of glycogen from fast twitch fibers against loads representing only 60% of maximum, it might be assumed that recruitment is not the sole neural mechanism used by muscles to maintain force.

MacDougall et al (1999) observed the effects of 1 or 3 sets to failure at 12 RM, in order to determine muscle substrate utilization over repetitive sets of resistance training. Intramuscular glycogen and creatine phosphate stores, in addition to the rate of glycogenolysis, were studied. Results indicated that glycogen was depleted by 12% and 24% after one and three sets, respectively. Conversely, creatine phosphate stores, although significantly decreased after sets one and three, were not different between the two conditions. The lack of differences were attributed to the ability of creatine phosphate to re-synthesize within the three-minute inter-set rest interval. However, during the three sets, MacDougall et al. observed an inability to perform the same number of repetitions on each set. Given, that creatine phosphate stores were similarly depleted after sets one and three and glycogenolysis continued to decrease, the researchers concluded that the rate of glycogenolysis effects the observed greater drop-off in repetitions performed during set 3 compared to set 1. Therefore, several metabolic systems, each of which require different amounts of time to recover, may contribute to fatigue during resistance training.

These observations are supported by Abdessemed, Duche, Hautier, Poumarat and Bedu (1999) who compared the power output during each repetition during a bench press

exercise in which the duration of rest intervals was different between protocols. Subjects performed 10 sets of 6 repetitions at 70% 1 RM utilizing either 1, 3 or 5 minute rest periods. Power output during each of the 6 repetitions per set was maintained over 10 sets in the 3 and 5 min rest conditions. Similar results were demonstrated in the 1 min rest protocol during the first 3 sets. However, from sets 4 to 10, a significant reduction in power output occurred during the final 3 repetitions per set, meaning that subjects in the 1 min rest group were able to maintain power outputs on the on the first three repetitions of each set. It is probable that the rate of delivery of high-energy phosphates enabled high-level short duration performance to continue over the first 3 repetitions and that performance decreased as a result of decreased glycogenolysis during repetitions 4-6. Given that 3 repetitions of resistance training at a controlled tempo would last approximately 6-12 s, these results are consistent with observations involving the timing of creatine phosphate depletion. Jones, McCartney, Graham et al. (1985) observed that creatine phosphate is near completely depleted approximately 10 s after the onset of high intensity muscle contractions

As a result of increased demands on the supply of ATP, it has been speculated that the by-products of hydrolysis during fatiguing protocols (i.e. Pi or lactic acid and hydrogen) prevent further muscle contractions from occurring, due to the prevention of E-C coupling at the sarcoplasmic reticulum.

Furthermore, the inability to completely recover from accrued fatigue over repetitive stresses may lead to increased muscle acidity. Research has shown that decreased pH, is a result of increased H^+ production, which is an indication of acidity within the involved muscle(s) and has been related to blood lactate concentrations

(McCartney et al., 1986). Decreases in pH influences one or all of the metabolic processes responsible for glycogenolysis, PCr re-synthesis, Ca^{2+} reabsorption, Ca^{2+} binding to troponin and subsequent cross bridge formation (McLester Jr, 1997). This decrease in pH has been shown to impair muscle performance (Cady et al. 1989). Similarly, high intensity exercise has been suggested to reduce the pH of muscle from 7.0 to 6.2 and concurrently increase in H^{+} resulting in inhibition of the contractile apparatus (Fitts & Balog, 1996)

Although, research is equivicol regarding the effects of glycogen reduction, it is obvious that metabolic adjustments occur, which directly or indirectly contributes to a muscle's ability to produce and maintain force. McCartney et al. (1986) examined glycogenolysis following repeated bouts of maximal cycle ergometry and found that both the rate of glycogenolysis and muscle lactate levels did not change from the third to the fourth bout when compared to the first. Similarly, the percent decrease of work from bout 1 decreased consistently during the first 3 bouts to a maximum level of 43.3% of bout 1, but remained the same for 4.

Other metabolites have been associated with blood lactate and hydrogen production, and therefore, may be involved in the processes leading to noticeable changes in force production. Evidence exists for failure at the level of the sarcoplasmic reticulum (Gollnick, Korge, Karpakka, & Saltin, 1991). This failure is likely influenced by calcium (Ca^{++}) and accumulating inorganic phosphate (Pi), such that increases in Pi occur in the sarcoplasmic reticulum where Pi binds with Ca^{++} to diminish the amount of CA^{++} released (Hepple, 2002). Furthemore, Ca^{++} seems to be affected by acidic environments, such that at a lowered pH, often associated with increased blood lactate results in an

increased Ca^{++} requirement for similar tension (Fabiato & Fabiato, 1978). Favero, Zable, Colter and Abrasom (1997) found that blood lactate inhibits Ca^{++} release from the sarcoplasmic reticulum, causing a decrease in Ca^{++} transients available to bind with troponin. Although this experiment was performed in single muscle cells in rabbits, the decrease in available Ca^{++} would likely disrupt E-C coupling preventing full tension development leading to muscle fatigue. Williams and Klug (1995) support these results, suggesting that control of intracellular Ca^{++} concentration may be important in determining the fatigue response in a muscle.

Similarly, Fitts (1994) suggested that under conditions simulating fatigue (i.e.increased blood lactate) that Ca^{++} resequestering by the sarcoplasmic reticulum becomes slowed, leading to alterations in the cross bridge kinetics of the muscle. These observations are supported by studies that have demonstrated that a decreased pH impairs Ca^{++} release from the sarcoplasmic reticulum (Cady et al. 1988).

However, other researchers have suggested that decreases in Ca^{++} resequestering and release only occurs during low intensity, long duration fatigue and the onset of fatigue occurs after the initiation of factors that are Ca^{++} independent (i.e. H^+ and P_i), which directly act at the cross bridge (Fitts & Balog, 1996). A recent review of inorganic phosphate's role in fatigue exist (Hepple, 2002), suggesting that Pi production affects cross bridge turnover rate. Furthermore, this turnover may be influenced by the availability of O_2. Observations that hypoxia increases both the rate of fatigue and accumulation of Pi provide evidence to support this contention (Hepple, 2002). Therefore, the effect of various by products on Ca^{++} release from the sarcoplasmic

reticulum must be further studied, especially during training protocols which heavily rely on glycolysis to supply ATP.

Conclusions regarding the influence of metabolic by-procucts on force reduction are affected by current research designs; most research has focused on the metabolic factors retroactive to the induced fatigue state, preventing a full understanding of the metabolic changes that occur at the same time as reductions in muscle force. Metabolite production occurs prior to and during several sets of resistance training protocols designed to elicit strength and hypertrophy gains. To fully understand the metabolic responses of neuromuscular fatigue, research designed to elucidate ongoing metabolic responses during fatigue must be carefully explored.

Motor Unit Recruitment

Prescription of moderate – high intensity fatiguing weight training protocols has resulted from research indicating that there is an orderly recruitment and derecruitment of muscle fibers, which reportedly follows the size principle (Deluca, Lefever, McCue, & Xenakis, 1982; Behm, 1995). The size principle suggests that type I fibers are recruited prior to type II fibers and has been supported by research involving resistance exercise carried to volitional fatigue. Milner and Brown (1973a) found that volitional fatigue lead to motor-unit activity from previously inactive type II fibers towards the end of a set. Specifically, slow twitch fibers, known to have low force generation capabilities compared to fast twitch fibers become fatigued, causing fast twitch fibers (high force generation) to become activated in order to maintain force output. Further, it is rationalized that to sufficiently stimulate adaptation you must not only stimulate fast

twitch fibers, but must apply an appropriate level of tension until activated fibers are derecruited. Fast twitch fibers are larger (McCall et al. 1996) and have a greater propensity for growth (Deluca, 1985). Therefore, strength and hypertrophic increases during experimental protocols have usually depended on momentary neuromuscular fatigue.

Momentary exhaustion using 80% of 1 RM has been suggested to occur when there is a 20% decrease in EMG activity (Macdougall, et al., 1999). Proponents of training to fatigue suggest that creating greater neuromuscular fatigue during resistance exercise, through the use of long, rather than short duration, high intensity protocols, increases the stimulus for developing strength and hypertrophy (Schott, McCully, & Rutherford, 1995), because more fibers become fully activated and deactivated (Rooney, Herbert & Belnave, 1994). Rooney, Herbert, and Belnave found that two protocols with significantly differing levels of fatigue produced different increases in strength over a six week program, utilizing 6 RM resistance. The protocol that induced the greatest levels of acute, accumulated fatigue based on the above arguments would lead to recruitment of the greatest number of available fibers and consequently the largest changes in 1 RM strength. The results supported this argument.

These arguments are not supported by research examining specific recruitment patterns of different muscles (Behm, 1995; Deluca et al. 1996). Wood, Lockwood and Creswell (1983) concluded that the recruitment of additional motor units likely does not occur above 75 percent of maximal voluntary contraction, suggesting that other mechanisms of neuromuscular control must contribute to the increases in force beyond this point, which is consistent with the observations of (Deluca et al. 1996).

Behm (1995) has suggested that any combination of increased firing frequency, recruitment, synchronization or synergist muscle activity, as well as reduced co-contraction of antagonist muscles may contribute to the maintenance of force output. Furthermore, the same researcher suggested that the pattern of EMG activity recorded during maximum voluntary contractions was not the same as EMG patterns associated with muscular fatigue. Similar results have been obtained during cycling activity. Green and Patla (1992) examined muscle activation of the triceps of subjects performing sprints on a cycle ergometer and found that the maintenance of high forces required for maximal efforts, required activation of all synergistic muscles.

In support of these results Behm (1995), Rutherford and Jones (1984) suggested that increases in muscle coordination are responsible for strength increases in the absence of myogenic adaptation. Synergistic muscle activity has been studied during forearm flexion in order to determine changes in muscle activity as prime muscles fatigue (Nakazawa, Kawakami, Fukunuga, Yano, & Miyashita, 1993). The results suggested the biceps brachii, the primary mover during elbow flexion was activated during the early phase of exercise, but prior to fatigue EMG readings shifted, such that an increase in electrical activity occurred in the brachiradialis, the primary synergist muscle utilized during forearm flexion. This response seems consistent regardless of the intensity of the resistance exercise. Tamaki, Kitada, Akamine, Murata, Sakou, and Kurata (1998) examined synergistic muscle actions between the medial and lateral gastrocnemius and soleus muscles during low level contractions. Results indicated that these muscles alternate the level of activity in order to maintain optimal force output. However, the fatiguing protocols represented only 10% of maximum voluntary contraction and lasted

for 210 min, preventing the extrapolation of these results to intense muscle contractions. Nonetheless, it seems plausible that the recruitment of synergistic muscle is necessary for the maintenance of force production.

Results from studies examining motor unit recruitment are difficult to interpret, as most studies examining recruitment have examined individual motor units at lower intensities than typically associated with strength and hypertrophy programs. Furthermore, such designs fail to integrate the complexities within the human neuromuscular system.

If central factors are considered independent of peripheral fatigue, it might be concluded that volitional exhaustion represents the inability of the neural system to maintain force at any given moment, to a set load / intensity, through a combination of reduced recruitment, decreased firing frequency and / or fatigue of the synergists and stabilizer muscles. In light of the previous arguments, increased fiber recruitment might not play as significant a role with resistances beyond 80% of 1 RM, especially in trained athletes.

In fact, many researchers have found that even untrained athletes can fully activate muscles during resistance training (Behm, 1995). It follows, that either strength training protocols which maximize acute neuromuscular fatigue may not produce the optimal stimulus for neuromuscular adaptation or that activation of neural mechanisms, in addition to recruitment, contribute to optimizing the stimulus for chronic adaptation. Furthermore, to justify the exclusive use of fatiguing protocols, activation of neural mechanisms other than motor unit recruitment must occur in order to maintain force, once the motor unit recruitment threshold of all fibers has been reached.

The Role of Fatigue in Resistance Training Protocols

Design of Training Protocols

Fleck and Kraemer (1998) have stated "the exercise protocol chosen may affect the results of the resistance program". However, experiments that have examined factors promoting neuromuscular adaptation have been equivocal. Long-term resistance training studies have produced significant chronic myogenic adaptation (Macdougall, Sale, Alway, & Sutton, 1984; McCall, Byrnes & Dickinson, 1996). However, this result has not been consistent (Alway, Grumbt, & Gonyea, 1988). Furthermore, the designs of the research protocols have varied considerably. Resistance training variables such as the length of study, training status of the subjects, the number of sets per exercise, repetitions per set, time under tension and rest intervals between sets, have not been consistent, making it difficult to compare the results from different studies.

In general, the recommendations for hypertrophy tend include shorter rest intervals (i.e. 30 – 120 s) and moderate to high volumes (Fleck & Kraemer, 1998). Studies have involved protocols ranging from 30 s to 3 min interset rest intervals, and 1-5 sets per exercise (Carpinelli & Otto, 1997). The number of repetitions/set determined to induce muscle fiber changes have also been variable. Repetitions as low as 4 per set (Chestnut & Docherty, 2000) and as high as 25 per set (Craig & Kang, 1995) have been reported to elicit chronic myogenic adaptation from resistance training.

High intensity, low volume protocols have been suggested to improve strength (Carpenelli & Otto, 1997). Current recommendations for strength training with minimum hypertrophy involve 3-5 min rest between sets (Fleck, & Kraemer, 1998). Research has

suggested that the greatest increases in strength occur when subjects train at high but submaximal loads (Dons, Bollerop, Bonde-Peterson, & Hancke, 1979; Behm, 1995), suggesting that intensity, rather than volume is more important to produce the largest increases in strength.

Both acute (Behm et al., 2002) and chronic (Chestnut & Docherty, 2000) neuromuscular responses to resistance training protocols utilizing different training intensities have been examined. Observations revealed no differences in the neuromuscular responses. However, both studies involved protocols in which the lower intensity groups performed a higher volume of work. Therefore, acute variables in addition to intensity may contribute the optimal chronic adaptation. In addition, research comparing the results of 1 set per exercise to 3 sets per exercise, have revealed similar strength increases over short training periods. The interactive roles of intensity and volume is supported by Goldberg, Etlinger, Goldspink and Jablecki (1975) who suggested that in order to provide the optimal stimulus, a threshold of muscular tension and volume must be met. These results suggest that when a sufficient stimulus is delivered to the neuromuscular system, such that the threshold of all available motor unit have been reached, other variables (i.e. volume, rest interval and failure) contribute to further neuromuscular fatigue. Furthermore, when training volume is sufficient, strength increases may be similar regardless of the variation in intensity (Baker et al., 1994).

Intensity and Volume

Both relative intensity and volume have been positively correlated with neurogenic (Robergs et al., 1991) and myogenic adaptations (Dudley, Tesch, Miller &

Buchanan. 1991). However, it is commonly believed that the primary stimulus for neurogenic changes is intensity (Moritani & DeVries, 1979; Atha, 1981) and a combination of volume and intensity is necessary in order to promote myogenic adaptations (Goldberg et al. 1975; Kraemer et al., 1998).

Indirect evidence for the importance of volume and intensity during resistance training emerges from research examining the effects of various loading protocols on acute hormonal release. Growth hormone (McCall et al., 1999) and testosterone (Hickson et al., 1994) have been related to changes in strength and hypertrophy. Consequently, it might be speculated that loading protocols most effective at eliciting this acute hormonal response are also best suited for inducing neuromuscular adaptation. Resistance training variables differentially affect post-exercise growth hormone (Gotshalk, Loebel, Nindl et al., 1997; Hakkinen and Pakarinen, 1993; Kraemer, Marchittal, Gordon et al., 1990; McCall et al., 1999) and testosterone (Hickson et al., 1994) levels. The association between GH and myogenic adaptation was supported by McCall et al. (1999). Results from the study indicated a .71 correlation between myogenic adaptation and acute growth hormone (GH) release over 12 weeks of training.

Furthermore, if changing the volume load, intensity, rest interval or the degree of muscular failure differentially affects the hormonal environment then it might be assumed that optimizing each of these variables will be the most effective protocol, especially as a potential mediator of neuromuscular adaptation.

Kraemer et al. (1990) examined how different resistance training protocols would affect the endogenous hormonal response under different volumes, intensities and rest intervals. Nine males were tested under six conditions. Half the conditions involved

protocols designed to promote hypertrophy; the other three conditions included protocols designed to induce strength adaptations. Comparisons were made between the primary hypertrophy sessions (10 RM load and 1 min inter-set rest intervals) and a session that manipulated training load (5 RM with 1 min inter-set rest intervals). As expected the primary hypertrophy session caused the greatest increase in GH post exercise. It is apparent from the data, changing one program variable (i.e. total volume) alters serum GH concentrations. Researchers also examined blood lactate levels after each protocol. The lactate levels of the subjects were highest during the hypertrophy loading protocol, suggesting that an inability to meet anaerobic metabolic demands may play a significant role in hormonal response. However, no systematic changes to blood lactate were observed. These observations extend the findings of VanHelder, Radomski, and Goode (1984), who provided evidence that changing various resistance training variables impacts the acute response of GH. However, the novelty of the stimulus or limited resistance training background of subjects in these studies may have effected the anabolic response.

Hakkinen and Pakarinen (1993) studied the response of 10 male experienced lifters completed after two very distinct but fatiguing exercise regimens. The same subjects performed each of the two sessions involving either 20 sets with 1 RM loads or 10 sets of 10 repetitions with10 RM loads (i.e. 70% 1 RM). The resistance was adjusted for each set, ensuring that maximal efforts were occurring at the end of each set. Results indicated significant differences in the acute release of both testosterone and GH between groups with the highest values measured after the 10 RM protocol. This occurred despite subjects resting 3 min between sets. Similarly, an increase in blood lactate was recorded

for both protocols and that release was positively correlated with the increase in GH. While the volume of the 10 RM protocol was similar to typical advanced bodybuilding workouts, the protocol differed from typical hypertrophy prescription by the manner in which the volume of exercise was distributed. Bodybuilders normally divide 10-12 sets for an individual body part across 3 or 4 exercises. These studies extended the results of Craig and Kang (1994), who observed that resistance training volume of exercise during resistance may contribute to acute GH response after resistance training protocols.

Craig and Kang (1994) compared acute growth hormone release before and after performing a back squat or a leg press involving different intensities (i.e. 75% or 90% of 1 RM). During leg press activity 10 RM loads elicited the greatest GH increase, whereas the 25 RM loads caused the greatest increase when the exercise protocol involved the back squat. Only the 10 and 25 RM workloads produced a significant increase in blood lactate in the back squat or leg press trials. The results indicate alterations acute resistance training variables affects GH levels. More importantly, these results also demonstrate that the volume of exercise significantly affects GH response to resistance exercise.

Similarly, when the rate of force development was examined, observations indicated higher volumes are more important than power output in causing the release of GH (Kraemer et al. 1990). Kraemer et al. examined the differential effects of volume and mean power output on post exercise GH response. Male weight trainers with 2 or more years experience with half squats performed three sets as follows: a single set involving 75 percent 1 RM, a single set involving 90 percent 1 RM, and a progressive protocol involving both a 75 percent and 90 percent set. The latter protocol examined total volume by allowing subjects to lift at their own pace until volitional fatigue, with loads and rest

periods controlled. The single-set protocol was designed to examine power output. Subjects performed as many lifts as possible in 15 sec, using the same workloads as in the progressive routine. Immediate post-exercise blood levels of human growth hormone were significantly elevated in the progressive protocol but not in the single-set protocol, regardless of exercise intensity employed. It was found that the progressive workout involved a greater volume of work but lower mean power output, whereas greater power was observed during the single set protocols. The results demonstrated that total volume during exercise had a more important influence on growth hormone response following exercise than did power output or intensity

Although, acute release of hormones like GH indicate a short-term physiological response to exercise, until this increased hormonal release can be causally related to long-term adaptation (i.e. hypertrophy), the results must be interpreted with care.

Few studies have directly compared the effect of different volumes of training over several weeks of training on GH and testoterone (T). However, Several research has looked at the importance of volume within a periodization model (Baker et al. 1994). Several studies have attempted to determine if the volume of resistance training affects the gains in performance (Baker et al. 1994; Ostrowski et al 1997; Starkey, Pollock, Ishida et al., 1996).

A review examining the benefit of 3 sets compared to single set protocols (Carpenilli & Otto, 1997). Although the results of the reviewed studies were equivocal, it was speculated that 3 set protocols offer no advantage over single set protocols. A closer examination reveals that most of these studies examined individuals with limited training backgrounds, perhaps leading researchers to the conclusion that low volume training was

as effective as high volume training in producing changes in strength and hypertrophy (Starkey et al., 1996). A recent study that examined performance gains over 20 weeks has helped to determine the time course of adaptation (Kramer et al. 1997). Results indicated that subjects involved in a single set protocol improved strength similarly to individuals performing 3 set protocols during the first 10 weeks. However, during the last 10 weeks the individuals performing 3 sets continued to improve strength at a faster rate than the single set. In contrast to strength increases muscle thickness, a measure of muscle hypertrophy was shown to be similar after 25 weeks of 1 or 3 sets of 8-12 repetitions involving 7 different resistance training exercises (Pollock, Abe, De Hoyas et al. 1998). Measurements occurred from 8 sites and revealed no differences in muscle thickness changes in either the lower or upper body. Muscle thickness measures do not accommodate for increases in fat deposition, preventing definitive conclusions that the higher volume group decreased intramuscular fat stores to a greater degree than the low volume protocol.

Ostrowski et al. (1997) conducted one of the few studies that have specifically examined the effect of volume manipulations on neuromuscular adaptation in trained individuals performing bench press and squat lifts. Male subjects performed 10 weeks of either 3, 6 or 12 sets of resistive exercise per muscle group. All groups performed the same protocol with the exception of volume. It was found that all groups increased the girth of all the muscles under investigation. More importantly, ultrasound indicated significant improvements without differences amongst the groups. Strength increases followed the same trend, in that subjects increased their bench press and squat lift regardless of the volume lifted. However, a closer examination of the data revealed that

the percent change in the rectus femoris for group 3 was nearly 3 times the percent change for the 6 set group and almost twice the 3 set group. Furthermore, by collapsing the data, analyzing only pre and post results the researchers inflated the power of the within subjects portion of the multivariate design. The decreased power of the between subjects (N=8) variable may have prevented the researchers from concluding that greater changes in girth occurred due to the increased volume.

Baker et al. (1994) examined the effects of three different protocols, involving similar volume and intensity over 12 weeks. Subjects with at least 6 months training experience performed 6 repetitions per set (Gp 1), 6.65 rep/set (Gp 2) or 6.27 repetitions per set (Gp 3). Maximal squat, bench press, and LBM significantly increased for all groups. The changes in strength significantly correlated with changes in LBM. Although this study does not permit any conclusions about the independent effects of volume and intensity, an appropriate volume and intensity are important for improvements in strength and hypertrophy.

Behm et al. (2002) utilized iEMG and interpolated twitch (ITT) to determine acute neuromuscular responses after performing one set of 5, 10 or 20 RM loading protocols. It was hypothesized that the 5 RM protocol should have produced the most inactivation. However, the similarities in inactivation may be related to an interaction of volume and intensity, as the 20 RM protocol would have involved a significantly greater volume of exercise. These results support the findings of Baker et al. (1994) observations, that both volume and intensity were involved in neuromuscular adaptations. Furthermore, if feedback from the periphery did occur, perhaps metabolic disturbances associated with higher volume protocols may have influenced the descending neural

drive. This observation was supported by the larger reductions of tetanic muscle twitch (Behm et al., 2002), suggesting that the 5 RM protocol did incur more neural fatigue but an interaction between peripheral and neural mechanisms prevented such a conclusion. It might be concluded from the above results that if exercise intensity is sufficient to appropriately stimulate the entire motor unit pool of involved muscles that total exercise volume determines the amount of neuromuscular fatigue.

Indirect evidence in support of the effect of volume on acute neuromuscular fatigue exists. Brandenburg et al.(2000) observed similar 20% reductions in iEMG and MVIC after acute forearm flexion exercise involving different intensities. Measurements of MVIC and iEMG were recorded before and after performing 3 sets or 6 sets of supported single arm elbow flexion at intensities representing approximately 77% of 1 RM and 85% 1 RM, respectively. Although, intensities were different, subjects were required to perform extra sets in the 6 RM protocol in order to equate volume and the time under tension of individuals involved in the 10 RM protocol. The lack of acute neuromuscular differences between the two loading conditions may be the result of similar training volumes. However, the training intensities utilized (77% and 85% of 1 RM) were very similar, possibly preventing significant acute differences between the 2 resistance protocols.

Further research in the area must be completed in order to determine the appropriate amount of volume and intensity needed to elicit the greatest increases in both neural and hypertrophic adaptations.

Rest Interval

Recent research suggests that typical strength programs do not allow sufficient rest for complete recuperation between sets, potentially reducing the total volume / workout (Pincervo et al., 1998; 1997). Furthermore, inadequate rest between sets has led to a drop-off of repetitions over the repetitive sets of resistance training performed at both 6 RM (Brandenburg et al., 2000) and 10 RM (Macdougall et al., 1999) intensities. In addition, recovery from protocols involving repetitions to failure during moderate to high intensity exercise has been shown to extend well past 3 min (Pincervo et al., 1997; MacDougall et al., 1999; Esposito, Orizio, & Veicsteinas., 1998). Furthermore, when sufficient rest is given during a bout of resistance training, it appears that only small decrements occur in performance over repetitive sets (Pincivero et al., 1997). Most research has indicated that longer rest intervals result in greater increases in strength (Robinson, Stone, Johnson, Penland, Warren, & Lewis, 1995; Pincivero et al., 1997; 1998). Robinson et al. found a significantly greater increase in 1 RM squat strength after five weeks of training in subjects using a three min rest interval as compared to those using a 30 sec rest period. However, the relative intensities and total volume of work were significantly different for each group, preventing conclusions based solely on the differences in rest intervals between sets.

In contrast (Kulling, Hardison, Jacobson, & Edwards 1998) examined increases in 1 RM leg and bench press strength after 12 weeks of compete body resistance training involving either 30 or 90 s inter-set rest. Results indicated a greater increase in strength with 30 s compared to 90 s inter-set rest . These results contraxt with the results if Kraemer et al. (1990, 1993) and Abdessmed et al. (1999), which demonstrated that blood

lactate was related to decreases in performance when short rest intervals were used. Kraemer et al. (1990) compared 10 RM protocols, where the only difference between sessions, designed to promote hypertrophy, was the amount of rest between sets. One group received 3 min rest between set whereas the other received 1 min rest prior to initiating the next set of the series. The volume load performed in each of the hypertrophy sessions was equated. It was concluded that when the rest period was shortened or the exercise duration increased, blood lactates also increased.

Rest intervals have also have been linked to a drop-off in the number of repetitions performed on sets of resistance training subsequent to set 1. Kraemer, Kilgore, Kraemer, and Castrcane (1992) used 2 min rest intervals between 3 sets of bench press, lat pull, leg extension and leg curl exercise performed at a 10 RM load. Subjects were relatively untrained but had past experience with resistance training. They experienced a significant drop-off in the number of repetitions performed on sets 2 and 3. The highest drop-off occurred as a result of performing upper body exercises (i.e. bench press and lat pulldown). These results indicated that the upper-body was more susceptible to the effects of neuromuscular fatigue than the lower body.

Failure

Failure has been defined as the inability to perform another repetition against a given load (Fleck & Kraemer, 1998; MacDougall, Ray, Sale, McCartney, Lee, & Garner, 1999) or as a temporary decrease in the maximum force generating capacity of muscle (Kent-Braun, 1999). During resistance training to failure, fatigue (metabolic and /or neural) begins at the onset of the first repetition and continues until an individual can no

longer maintain sufficient force to complete another repetition. Therefore, protocols that involve repetitions to failure will induce the greatest amount of fatigue. Current theories suggest that heavy resistances or more appropriately the relative intensity (% 1RM) may be the more important factor during resistance training designed to promote chronic myogenic and neurogenic adaptation (Atha, 1981, Dons et al., 1979).

However, results of research have been equivocal and, therefore, researchers have had difficulty arriving at a consensus regarding the ideal protocol to induce performance changes. In general, a variety of resistance training programs using progressive overload have resulted in similar chronic neurogenic and myogenic adaptations. Variables such as repetitions to failure, total volume, rest interval and intensity all contribute to the level of acute neuromuscular fatigue and, therefore, may effect the desired outcome and should therefore be considered when designing resistance training protocols.

Few studies have examined and compare protocols involving repetitions to failure (or beyond) compared to not performing repetitions to failure during resistance training. (Stowers, Mcmillian, Scala, Davis, Wilson, & Stone, 1983; Stussi, Freitag, Hauenstein, Wylder, Eiganmann, & Boutellier, 1998; Nimmons, Marsit, Conley, Johnson, Honeycutt, & Hoke 1995; Berger & Hardage, 1967).

Results from these studies are relatively difficult to interpret because of the different volume of exercise performed by comparison groups (i.e. one set vs. multiple sets). Berger and Hardage's (1967) study highlights this methodological flaw. The researchers compared strength increases produced by bench press training protocols utilizing either 10 repetitions at 10 RM loads or 10 maximal contractions (i.e. 1 RM). In the latter, the load was reduced each time the subject could not perform the lift on his

own. Training was completed three times a week for 8 weeks. Subjects who trained with maximal loads on every repetition incurred a 60 % greater increase in 1 RM bench press. Berger and Hardage argued that the greater increase in strength could not be attributed to greater training intensity during the maximal contraction protocol because the mean training load lifted was less in the maximal contraction group. Therefore, results were attributed to a greater level of failure and fatigue induced by repeated maximal efforts. However, the mechanical stress occurring during the first 4 or 5 repetitions would not have been experienced by the 10 RM group. Perhaps contribution of the larger initial training loads utilized in the maximal protocol increased the involvement of neural strategies or greater myofibril disruption than the 10 RM protocol.

The role of repetitions to failure towards promoting neuromuscular adaptation is not scientifically well understood. However, research has compared the differences between bodybuilders and strength/power athletes (MacDougall et al., 1994; Tesch & larson, 1982). While bodybuilding protocols often involve repetitions to failure, powerlifters seldom perform a failed repetition during resistance training. Tesch and Larson compared the muscle fiber constitution of a group of competitive bodybuilders to a group of competitive weight/power-lifters. Muscle biopsies were obtained from the vastus lateralis and medial deltoidius. Tissue samples were analyzed with respect to the relative distribution of fast and slow twitch fibers as well individual fiber areas. Measurements indicated mean fiber area was similar in the vastus lateralis of both groups and smaller in the medial deltoidius of bodybuilders. Although this study did not examine potential hyperplasia, perhaps greater individual fiber hypertrophy does not result from inducing repetitive failure. However, Fry, Kraemer, Triplett, and Knuttgen

(1995) similarly concluded, although there can be differences in the size and number of fiber types, the average cell size among the three groups was not different.

Research exists that has found the training protocols involving repetitions to failure had greater chronic increases in neurogenic (Rooney et al., 1994) and myogeinc adaptation (Schott et al., 1995). Schott et al. compared the effects of 14 weeks of continuous isometric training with a protocol involving intermittent isometric training. Both protocols involved an intensity representing 70% of MVC. The continuous group performed 4 isometric contractions involving 30-second duration, while the intermittent group performed 4 sets of 10 isometric contractions, utilizing a 3 s on 3 s off format. Acute metabolic changes (pH and Pi:PCr ratio) within the muscle, measured during initial training sessions, were larger in the continuous condition although the intensity and total exercise duration were equivalent to the intermittent protocol. The continuous contraction group had significantly greater Myofibrillar and strength increases (54.7% versus 31.5%). Although the researchers suggested that the greater fluctuations in metabolic by products likely caused the increased gains, it is possible that the recruitment strategies and patterns of a long contraction is different from short contractions, preventing conclusions regarding the cause of the observed neuromuscular increases.

Experimental evidence supporting repetitions to failure as the optimal training protocol is limited and there is research that supports training protocols that do not involve training to failure on each set (Stussi, et al., 1998). Stussi et al. is one of few studies that has examined repetitions to failure in a manner representative of typical strength training. Twenty-three males were tested for 1 RM prior to and after performing 24 sessions (3x per week) of leg press and back extension exercises. Subjects performed

either 15 repetitions to failure at a 15 RM load or 2 sets of 6 repetitions at a 10 RM load (non-failure). The rest interval was 2 min between sets. It was found that the non-failure group increased 1 RM similarly to the group the went to failure. Alhough this study equated total volume (34359 ± 7071) and (32026 ± 7819) for the leg press, relative intensities were different between groups and may have contributed to the strength gains. It is conceivable that mechanical tension evoked from a 10 RM load may be significantly different, especially if the stimulus for strength increase is intensity. The results from the aforementioned studies suggest that training to failure (or beyond) offers no particular advantage, which is consistent with recent reviews (Baker et al. 1998; Stone et al., 1996). Additionally the work of Nimmons et al. (1995), suggests that consistently training to volitional exhaustion may lead to overtraining. Recent periodized programs for strength seem to reflect these sentiments (Baker, 1998).

Conclusion

The mechanisms surrounding fatigue are complex, thus the ability of fatiguing contractions to facilitate training increases in strength and hypertrophy are unclear (Rooney et al., 1994). However, it appears that the acute changes to resistance training variables influences physiological outcome. Perhaps, the best program for increasing neuromuscular adaptation within muscle, is a program that optimizes the intensity, and volume of resistance training performed by manipulating inter-set rest intervals and the degree of neuromuscular fatigue. In light of current hormone research a program designed to optimize but not maximizing each training variable could theoretically induce

the largest neurogenic and myogenic adaptations. Research has indicated that the percentage of 1 RM required to cause significant type IIa and type IIb fiber specific glycogen depletion is much lower than current recommendations for training. It may be inferred that motor-unit recruitment occurs with training loads/intensities much less than typically used in experiments that have induced increases in strength or hypertrophy (Tesch et al., 1998; Robergs et al., 1991). If full motor unit recruitment is occurring at lower intensities perhaps other neuromuscular mechanisms are partially responsible for changes in muscle performance. Other mechanisms could include synergistic muscle activity, less coactivation of the antagonist, and changes in the biochemical composition of the trained muscles. Complete exploration of these mechanisms will be necessary to help the understanding of the processes involved in muscle cell growth.

Although, volume and intensity appear to contribute to chronic adaptation, research seems to indicate that performing repetitions to failure, especially in combination with inadequate interset rest interval may be counterproductive towards maximizing either volume or intensity during an acute bout of resistance training. Therefore, chronic neuromuscular adaptation to moderate and high intensity submaximal resistance training protocols might be compromised under the aforementioned conditions.

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Appendix C: Informed Consent

CONSENT FORM

You are being asked to voluntarily participate in a project entitled "exhaustion versus non-exhaustion during intermittent sub-maximal resistance training" that is being conducted by graduate students, Chad Benson, and Jason Brandenburg as part of the requirements for the Master's and Doctorate degrees in Exercise and Sport Studies at the University of Victoria. If you have any questions or concerns about the project, you may contact either the student at 386-4509 or call his/her graduate supervisor, Dr. David Docherty at 721- 8373.

The purpose of this project is to determine if blood lactates and integrate electromyographic responses during resistance training are dependent upon acute volitional exhaustion. In addition, the researchers will examine if this exhaustive state is the same after performing low (i.e. 1-4) and moderate (i.e. 10-12) repetitions.

The resources for conducting this study are being provided by the experimenters. The experimenters have solicited, and require the use of the Sport and Fitness Centre at the University of Victoria. A verbal agreement with the immediate weight-room supervisor has been obtained.

The benefits of participating in this study include closely supervised resistance training sessions by a qualified personal trainer/strength and conditioning coach. Participants are expected to gain knowledge of proper exercise technique and weight-room protocols.

The costs/inconvenience of participating include finger pricks, the individuals personal time, and possible, but minimal delayed onset muscle soreness. The purpose of the finger prick is so a small (droplet) blood sample can be taken and analyzed for lactate levels. Because an auto-lancet is utilized in this finger prick technique it is considered invasive.

If you agree to participate you will be asked to be part of a pilot study, which will help develop the protocols to be used in the research study. Involvement will initially include an orientation and question session. During the orientation all exercise techniques will be reviewed and feedback given. This will be followed by strength testing for individual 1 and 10 repetition maximums (i.e. the maximum weight an individual can lift 1 and10 times respectively before temporary exhaustion). At orientation end, any or all questions surrounding the preliminary study will be answered.
The experimental protocol will require each subject to perform a maximum 8 strength training sessions over 2 weeks. Four of the sessions will involve 3 sets of ten repetitions to exhaustion, utilizing a maximum of 3 different exercises. The other four sessions will involve 5 sets of 1-6 repetitions, again utilizing a maximum of 3 different exercises. To ensure the performance of proper technique, both researchers will supervise all sessions. The researcher and the subjects themselves on personal training logs provided by the investigator will record all data. Prior to and upon completion of each exercise session blood samples and surface electromyographic recordings will be performed.

Your participation in this project is entirely voluntary and you are free to refuse to participate, to withdraw from it, or to refuse to answer certain questions, without any negative consequences. In the event that you withdraw from the study, your data will be made available to you for future reference. Otherwise it will be destroyed.

Your anonymity and confidentiality will be protected by placing all obtained information in a locked cabinet at my home. All subjects will also keep a personal journal for their own personal use. The researcher will refer to all subjects and their data on a numerical system starting at 1. Only the experimenters will handle the information, but a spare key, in case of loss will be left with the supervisor. At the conclusion of the study, all of the raw data will be kept under lock and key in our personal archives. Data collection procedures and access to data for analysis will be limited to the principal investigator (Chad Benson), the co-investigator (Jason Brandenburg) and the supervisor Dr. Dave Docherty.

The results of this study are only preliminary and will be prepared for presentation at a special meeting with the supervisor. In addition, the preliminary results will be made available to all subjects. Having understood the above information and been given an opportunity to have my questions answered, I agree to participate in this study:

Signature of Participant _____
Signature of Witness_____

Appendix D: SPSS 9.0 Statistical Analysis

-Test

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	SET1A	10.4615	13	1.39137	.38590
	SET1B	9.9231	13	.27735	.07692
Pair 2	SET2A	7.7692	13	1.23517	.34257
	SET2B	9.9231	13	.27735	.07692
Pair 3	SET3A	6.0769	13	1.55250	.43059
	SET3B	10.6154	13	1.66024	.46047

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	SET1A & SET1B	13	.100	.746
Pair 2	SET2A & SET2B	13	-.299	.320
Pair 3	SET3A & SET3B	13	.077	.802

Paired Samples Test

		Paired Differences					t
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper	
Pair 1	SET1A - SET1B	.5385	1.39137	.38590	-.3023	1.3793	1.395
Pair 2	SET2A - SET2B	-2.1538	1.34450	.37290	-2.9663	-1.3414	-5.776
Pair 3	SET3A - SET3B	-4.5385	2.18386	.60569	-5.8582	-3.2188	-7.493

Paired Samples Test

		df	Sig. (2-tailed)
Pair 1	SET1A - SET1B	12	.188
Pair 2	SET2A - SET2B	12	.000
Pair 3	SET3A - SET3B	12	.000

-Test

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	VOLCHADA	901.7231	13	115.5829	32.0569
	VOLBCHAD	1028.6077	13	147.7370	40.9749
Pair 2	AMVICPRE	88.8455	11	9.8282	2.9633
	BMVICPRE	89.0818	11	9.3953	2.8328
Pair 3	AMVICPRE	89.1917	12	9.4472	2.7272
	AMVICPOS	73.2917	12	9.2903	2.6819
Pair 4	BMVICPRE	88.0250	12	9.6773	2.7936
	BMVICPOS	73.0233	12	12.0374	3.4749
Pair 5	ALACPRE	1.3385	13	.4011	.1113
	BLACBPRE	1.7462	13	.6319	.1753
Pair 6	ALACPRE	1.3385	13	.4011	.1113
	ALACPOST	2.9231	13	.6845	.1899
Pair 7	ALACPOST	2.9231	13	.6845	.1899
	BLACPOST	2.7615	13	.7633	.2117
Pair 8	BLACBPRE	1.7462	13	.6319	.1753
	BLACPOST	2.7615	13	.7633	.2117
Pair 9	EMGPRE	2.6238	13	.9434	.2617
	EMGBPRE	3.5860	13	2.1187	.5876
Pair 10	EMGPRE	2.6238	13	.9434	.2617
	EMGPOST	2.1844	13	1.3467	.3735
Pair 11	EMGBPRE	3.5860	13	2.1187	.5876
	EMGBPOST	2.9368	13	1.8517	.5136
Pair 12	EMGPOST	2.1844	13	1.3467	.3735
	EMGBPOST	2.9368	13	1.8517	.5136
Pair 13	AREMGBPR	.4009	13	.2724	7.556E-02
	AREMGAPR	.2058	13	.1280	3.551E-02
Pair 14	AREMGAPR	.2058	13	.1280	3.551E-02
	AREMGAPO	.1918	13	.1462	4.056E-02
Pair 15	AREMGBPR	.4009	13	.2724	7.556E-02
	AREMGBPO	.2657	13	.2164	6.002E-02

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	VOLCHADA & VOLBCHAD	13	.650	.016
Pair 2	AMVICPRE & BMVICPRE	11	.665	.026
Pair 3	AMVICPRE & AMVICPOS	12	.823	.001
Pair 4	BMVICPRE & BMVICPOS	12	.936	.000
Pair 5	ALACPRE & BLACBPRE	13	-.583	.037
Pair 6	ALACPRE & ALACPOST	13	.285	.346
Pair 7	ALACPOST & BLACPOST	13	.002	.995
Pair 8	BLACBPRE & BLACPOST	13	.453	.120
Pair 9	EMGPRE & EMGBPRE	13	.472	.104
Pair 10	EMGPRE & EMGPOST	13	.883	.000
Pair 11	EMGBPRE & EMGBPOST	13	.987	.000
Pair 12	EMGPOST & EMGBPOST	13	.523	.067
Pair 13	AREMGBPR & AREMGAPR	13	.617	.025
Pair 14	AREMGAPR & AREMGAPO	13	.236	.438
Pair 15	AREMGBPR & AREMGBPO	13	.551	.051

Paired Samples Test

		Paired Differences					t
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper	
Pair 1	VOLCHADA - VOLBCHAD	-126.8846	113.9498	31.6040	-195.7438	-58.0255	-4.015
Pair 2	AMVICPRE - BMVICPRE	-.2364	7.8761	2.3747	-5.5276	5.0549	-.100
Pair 3	AMVICPRE - AMVICPOS	15.9000	5.5821	1.6114	12.3533	19.4467	9.867
Pair 4	BMVICPRE - BMVICPOS	15.0017	4.5233	1.3058	12.1277	17.8756	11.489
Pair 5	ALACPRE - BLACBPRE	-.4077	.9251	.2566	-.9667	.1513	-1.589
Pair 6	ALACPRE - ALACPOST	-1.5846	.6878	.1908	-2.0003	-1.1690	-8.307
Pair 7	ALACPOST - BLACPOST	.1615	1.0243	.2841	-.4575	.7805	.569
Pair 8	BLACBPRE - BLACPOST	-1.0154	.7381	.2047	-1.4614	-.5694	-4.960
Pair 9	EMGPRE - EMGBPRE	-.9622	1.8688	.5183	-2.0915	.1672	-1.856
Pair 10	EMGPRE - EMGPOST	.4395	.6789	.1883	2.919E-02	.8497	2.334
Pair 11	EMGBPRE - EMGBPOST	.6492	.4163	.1155	.3976	.9008	5.622
Pair 12	EMGPOST - EMGBPOST	-.7525	1.6232	.4502	-1.7333	.2284	-1.671
Pair 13	AREMGBPR - AREMGAPR	.1951	.2180	6.047E-02	6.332E-02	.3268	3.226
Pair 14	AREMGAPR - AREMGAPO	1.408E-02	.1702	4.719E-02	-8.87E-02	.1169	.298
Pair 15	AREMGBPR - AREMGBPO	.1352	.2369	6.569E-02	-7.90E-03	.2784	2.059

Paired Samples Test

		df	Sig. (2-tailed)
Pair 1	VOLCHADA - VOLBCHAD	12	.002
Pair 2	AMVICPRE - BMVICPRE	10	.923
Pair 3	AMVICPRE - AMVICPOS	11	.000
Pair 4	BMVICPRE - BMVICPOS	11	.000
Pair 5	ALACPRE - BLACBPRE	12	.138
Pair 6	ALACPRE - ALACPOST	12	.000
Pair 7	ALACPOST - BLACPOST	12	.580
Pair 8	BLACBPRE - BLACPOST	12	.000
Pair 9	EMGPRE - EMGBPRE	12	.088
Pair 10	EMGPRE - EMGPOST	12	.038
Pair 11	EMGBPRE - EMGBPOST	12	.000
Pair 12	EMGPOST - EMGBPOST	12	.120
Pair 13	AREMGBPR - AREMGAPR	12	.007
Pair 14	AREMGAPR - AREMGAPO	12	.771
Pair 15	AREMGBPR - AREMGBPO	12	.062

·Test

Paired Samples Statistics

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	AMVICPOS	71.8000	13	10.39439	2.88288
	BMVICPOS	73.5215	13	11.66406	3.23503
Pair 2	AREMGBPO	.2657	13	.21642	.06002
	AREMGAPO	.1918	13	.14622	.04056

Paired Samples Correlations

		N	Correlation	Sig.
Pair 1	AMVICPOS & BMVICPOS	13	.741	.004
Pair 2	AREMGBPO & AREMGAPO	13	.407	.168

Paired Samples Test

		Paired Differences					t
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference		
					Lower	Upper	
Pair 1	AMVICPOS - BMVICPOS	-1.7215	8.02986	2.22708	-6.5739	3.1309	-.773
Pair 2	AREMGBPO - AREMGAPO	.0739	.20612	.05717	-.0506	.1985	1.293

Paired Samples Test

		df	Sig. (2-tailed)
Pair 1	AMVICPOS - BMVICPOS	12	.454
Pair 2	AREMGBPO - AREMGAPO	12	.220

ieneral Linear Model

Within-Subjects Factors

Measure: MEASURE_1

EMGA	EMGB	Dependent Variable
1	1	EMGPRE
	2	EMGPOST
2	1	EMGBPRE
	2	EMGBPOST

Descriptive Statistics

	Mean	Std. Deviation	N
EMGPRE	2.6238	.9434	13
EMGPOST	2.1844	1.3467	13
EMGBPRE	3.5860	2.1187	13
EMGBPOST	2.9368	1.8517	13

Multivariate Tests[c]

Effect		Value	F	Hypothesis df	Error df	Sig.	Eta Squared
EMGA	Pillai's Trace	.217	3.321[b]	1.000	12.000	.093	.217
	Wilks' Lambda	.783	3.321[b]	1.000	12.000	.093	.217
	Hotelling's Trace	.277	3.321[b]	1.000	12.000	.093	.217
	Roy's Largest Root	.277	3.321[b]	1.000	12.000	.093	.217
EMGB	Pillai's Trace	.711	29.547[b]	1.000	12.000	.000	.711
	Wilks' Lambda	.289	29.547[b]	1.000	12.000	.000	.711
	Hotelling's Trace	2.462	29.547[b]	1.000	12.000	.000	.711
	Roy's Largest Root	2.462	29.547[b]	1.000	12.000	.000	.711
EMGA * EMGB	Pillai's Trace	.060	.765[b]	1.000	12.000	.399	.060
	Wilks' Lambda	.940	.765[b]	1.000	12.000	.399	.060
	Hotelling's Trace	.064	.765[b]	1.000	12.000	.399	.060
	Roy's Largest Root	.064	.765[b]	1.000	12.000	.399	.060

Multivariate Tests[c]

Effect		Noncent. Parameter	Observed Power[a]
EMGA	Pillai's Trace	3.321	.389
	Wilks' Lambda	3.321	.389
	Hotelling's Trace	3.321	.389
	Roy's Largest Root	3.321	.389
EMGB	Pillai's Trace	29.547	.999
	Wilks' Lambda	29.547	.999
	Hotelling's Trace	29.547	.999
	Roy's Largest Root	29.547	.999
EMGA * EMGB	Pillai's Trace	.765	.127
	Wilks' Lambda	.765	.127
	Hotelling's Trace	.765	.127
	Roy's Largest Root	.765	.127

a. Computed using alpha = .05

b. Exact statistic

c.
Design: Intercept
Within Subjects Design: EMGA+EMGB+EMGA*EMGB

Mauchly's Test of Sphericity[b]

Measure: MEASURE_1

Within Subjects Effect	Mauchly's W	Approx. Chi-Square	df	Sig.	Epsilon[a]		
					Greenhouse-Geisser	Huynh-Feldt	Lower-bound
EMGA	1.000	.000	0	.	1.000	1.000	1.000
EMGB	1.000	.000	0	.	1.000	1.000	1.000
EMGA * EMGB	1.000	.000	0	.	1.000	1.000	1.000

Tests the null hypothesis that the error covariance matrix of the orthonormalized transformed dependent variables is proportional to an identity matrix.

a. May be used to adjust the degrees of freedom for the averaged tests of significance. Corrected tests are displayed in the Tests of Within-Subjects Effects table.

b.
Design: Intercept
Within Subjects Design: EMGA+EMGB+EMGA*EMGB

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Type III Sum of Squares	df	Mean Square	F	Sig.
EMGA	Sphericity Assumed	9.555	1	9.555	3.321	.093
	Greenhouse-Geisser	9.555	1.000	9.555	3.321	.093
	Huynh-Feldt	9.555	1.000	9.555	3.321	.093
	Lower-bound	9.555	1.000	9.555	3.321	.093
Error(EMGA)	Sphericity Assumed	34.521	12	2.877		
	Greenhouse-Geisser	34.521	12.000	2.877		
	Huynh-Feldt	34.521	12.000	2.877		
	Lower-bound	34.521	12.000	2.877		
EMGB	Sphericity Assumed	3.852	1	3.852	29.547	.000
	Greenhouse-Geisser	3.852	1.000	3.852	29.547	.000
	Huynh-Feldt	3.852	1.000	3.852	29.547	.000
	Lower-bound	3.852	1.000	3.852	29.547	.000
Error(EMGB)	Sphericity Assumed	1.564	12	.130		
	Greenhouse-Geisser	1.564	12.000	.130		
	Huynh-Feldt	1.564	12.000	.130		
	Lower-bound	1.564	12.000	.130		
EMGA * EMGB	Sphericity Assumed	.143	1	.143	.765	.399
	Greenhouse-Geisser	.143	1.000	.143	.765	.399
	Huynh-Feldt	.143	1.000	.143	.765	.399
	Lower-bound	.143	1.000	.143	.765	.399
Error(EMGA*EMGB)	Sphericity Assumed	2.241	12	.187		
	Greenhouse-Geisser	2.241	12.000	.187		
	Huynh-Feldt	2.241	12.000	.187		
	Lower-bound	2.241	12.000	.187		

Tests of Within-Subjects Effects

Measure: MEASURE_1

Source		Eta Squared	Noncent. Parameter	Observed Power[a]
EMGA	Sphericity Assumed	.217	3.321	.389
	Greenhouse-Geisser	.217	3.321	.389
	Huynh-Feldt	.217	3.321	.389
	Lower-bound	.217	3.321	.389
Error(EMGA)	Sphericity Assumed			
	Greenhouse-Geisser			
	Huynh-Feldt			
	Lower-bound			
EMGB	Sphericity Assumed	.711	29.547	.999
	Greenhouse-Geisser	.711	29.547	.999
	Huynh-Feldt	.711	29.547	.999
	Lower-bound	.711	29.547	.999
Error(EMGB)	Sphericity Assumed			
	Greenhouse-Geisser			
	Huynh-Feldt			
	Lower-bound			
EMGA * EMGB	Sphericity Assumed	.060	.765	.127
	Greenhouse-Geisser	.060	.765	.127
	Huynh-Feldt	.060	.765	.127
	Lower-bound	.060	.765	.127
Error(EMGA*EMGB)	Sphericity Assumed			
	Greenhouse-Geisser			
	Huynh-Feldt			
	Lower-bound			

a. Computed using alpha = .05

Tests of Within-Subjects Contrasts

Measure: MEASURE_1

Source	EMGA	EMGB	Type III Sum of Squares	df	Mean Square	F	Sig.
EMGA	Linear		9.555	1	9.555	3.321	.093
Error(EMGA)	Linear		34.521	12	2.877		
EMGB		Linear	3.852	1	3.852	29.547	.000
Error(EMGB)		Linear	1.564	12	.130		
EMGA * EMGB	Linear	Linear	.143	1	.143	.765	.399
Error(EMGA*EMG	Linear	Linear	2.241	12	.187		

Tests of Within-Subjects Contrasts

Measure: MEASURE_1

Source	EMGA	EMGB	Eta Squared	Noncent. Parameter	Observed Power[a]
EMGA	Linear		.217	3.321	.389
Error(EMGA)	Linear				
EMGB		Linear	.711	29.547	.999
Error(EMGB)		Linear			
EMGA * EMGB	Linear	Linear	.060	.765	.127
Error(EMGA*EMG	Linear	Linear			

a. Computed using alpha = .05

Tests of Between-Subjects Effects

Measure: MEASURE_1
Transformed Variable: Average

Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Eta Squared	Noncent. Parameter	Observed Power[a]
Intercept	417.278	1	417.278	56.183	.000	.824	56.183	1.000
Error	89.126	12	7.427					

a. Computed using alpha = .05

General Linear Model

Within-Subjects Factors

Measure: MEASURE_1

MVICA	MVICB	Dependent Variable
1	1	AMVICPRE
	2	AMVICPOS
2	1	BMVICPRE
	2	BMVICPOS

Descriptive Statistics

	Mean	Std. Deviation	N
AMVICPRE	88.8455	9.8282	11
AMVICPOS	72.6091	9.4229	11
BMVICPRE	89.0818	9.3953	11
BMVICPOS	74.6255	11.2030	11

orrelations

Correlations

		VOLUMEA	VOLBCHAD	AMVICPOS	BMVICPOS	ALACPOST
VOLUMEA	Pearson Correlation	1.000	-.405	-.338	-.270	.003
	Sig. (2-tailed)	.	.191	.283	.396	.993
	N	12	12	12	12	12
VOLBCHAD	Pearson Correlation	-.405	1.000	.571*	.736**	-.018
	Sig. (2-tailed)	.191	.	.042	.004	.953
	N	12	13	13	13	13
AMVICPOS	Pearson Correlation	-.338	.571*	1.000	.741**	-.223
	Sig. (2-tailed)	.283	.042	.	.004	.465
	N	12	13	13	13	13
BMVICPOS	Pearson Correlation	-.270	.736**	.741**	1.000	-.315
	Sig. (2-tailed)	.396	.004	.004	.	.294
	N	12	13	13	13	13
ALACPOST	Pearson Correlation	.003	-.018	-.223	-.315	1.000
	Sig. (2-tailed)	.993	.953	.465	.294	.
	N	12	13	13	13	13
BLACPOST	Pearson Correlation	-.572	.007	.061	.026	.002
	Sig. (2-tailed)	.052	.982	.844	.933	.995
	N	12	13	13	13	13
EMGPOST	Pearson Correlation	.015	.455	.513	.367	.220
	Sig. (2-tailed)	.964	.118	.073	.217	.471
	N	12	13	13	13	13
EMGBPOST	Pearson Correlation	-.105	.092	.286	.198	.106
	Sig. (2-tailed)	.745	.766	.344	.517	.731
	N	12	13	13	13	13
AREMGBPO	Pearson Correlation	-.024	.235	.319	.263	.243
	Sig. (2-tailed)	.940	.440	.288	.385	.425
	N	12	13	13	13	13
AREMGAPO	Pearson Correlation	-.260	.336	.132	-.020	.572*
	Sig. (2-tailed)	.414	.261	.667	.947	.041
	N	12	13	13	13	13
MASS	Pearson Correlation	-.534	.432	.677*	.381	-.167
	Sig. (2-tailed)	.074	.141	.011	.199	.587
	N	12	13	13	13	13
AGE	Pearson Correlation	.010	.171	.195	-.021	-.183
	Sig. (2-tailed)	.976	.576	.524	.945	.550
	N	12	13	13	13	13

Correlations

		BLACPOST	EMGPOST	EMGBPOST	AREMGBPO	AREMGAPO
VOLUMEA	Pearson Correlation	-.572	.015	-.105	-.024	-.260
	Sig. (2-tailed)	.052	.964	.745	.940	.414
	N	12	12	12	12	12
VOLBCHAD	Pearson Correlation	.007	.455	.092	.235	.336
	Sig. (2-tailed)	.982	.118	.766	.440	.261
	N	13	13	13	13	13
AMVICPOS	Pearson Correlation	.061	.513	.286	.319	.132
	Sig. (2-tailed)	.844	.073	.344	.288	.667
	N	13	13	13	13	13
BMVICPOS	Pearson Correlation	.026	.367	.198	.263	-.020
	Sig. (2-tailed)	.933	.217	.517	.385	.947
	N	13	13	13	13	13
ALACPOST	Pearson Correlation	.002	.220	.106	.243	.572*
	Sig. (2-tailed)	.995	.471	.731	.425	.041
	N	13	13	13	13	13
BLACPOST	Pearson Correlation	1.000	-.300	-.355	-.274	-.002
	Sig. (2-tailed)	.	.319	.235	.365	.994
	N	13	13	13	13	13
EMGPOST	Pearson Correlation	-.300	1.000	.523	.758**	.624*
	Sig. (2-tailed)	.319	.	.067	.003	.023
	N	13	13	13	13	13
EMGBPOST	Pearson Correlation	-.355	.523	1.000	.906**	.204
	Sig. (2-tailed)	.235	.067	.	.000	.504
	N	13	13	13	13	13
AREMGBPO	Pearson Correlation	-.274	.758**	.906**	1.000	.407
	Sig. (2-tailed)	.365	.003	.000	.	.168
	N	13	13	13	13	13
AREMGAPO	Pearson Correlation	-.002	.624*	.204	.407	1.000
	Sig. (2-tailed)	.994	.023	.504	.168	.
	N	13	13	13	13	13
MASS	Pearson Correlation	.108	.508	.649*	.623*	.169
	Sig. (2-tailed)	.726	.076	.016	.023	.582
	N	13	13	13	13	13
AGE	Pearson Correlation	-.397	.064	.283	.183	.091
	Sig. (2-tailed)	.179	.836	.349	.549	.768
	N	13	13	13	13	13

Correlations

		MASS	AGE
VOLUMEA	Pearson Correlation	-.534	.010
	Sig. (2-tailed)	.074	.976
	N	12	12
VOLBCHAD	Pearson Correlation	.432	.171
	Sig. (2-tailed)	.141	.576
	N	13	13
AMVICPOS	Pearson Correlation	.677*	.195
	Sig. (2-tailed)	.011	.524
	N	13	13
BMVICPOS	Pearson Correlation	.381	-.021
	Sig. (2-tailed)	.199	.945
	N	13	13
ALACPOST	Pearson Correlation	-.167	-.183
	Sig. (2-tailed)	.587	.550
	N	13	13
BLACPOST	Pearson Correlation	.108	-.397
	Sig. (2-tailed)	.726	.179
	N	13	13
EMGPOST	Pearson Correlation	.508	.064
	Sig. (2-tailed)	.076	.836
	N	13	13
EMGBPOST	Pearson Correlation	.649*	.283
	Sig. (2-tailed)	.016	.349
	N	13	13
AREMGBPO	Pearson Correlation	.623*	.183
	Sig. (2-tailed)	.023	.549
	N	13	13
AREMGAPO	Pearson Correlation	.169	.091
	Sig. (2-tailed)	.582	.768
	N	13	13
MASS	Pearson Correlation	1.000	.296
	Sig. (2-tailed)	.	.325
	N	13	13
AGE	Pearson Correlation	.296	1.000
	Sig. (2-tailed)	.325	.
	N	13	13

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

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Title of Thesis

Acute Neuromuscular Responses to Resistance Training Performed at 100% of 10 RM and 90% of 10 RM

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